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Issue Date: 06 March 2007

Case No.: 2006-BLA-5465
2006-BLA-5466

In the Matter of:

M. F. A., widow of and o/b/o
L. S. A. (D),
Claimant

v.

Peerless Eagle Coal Company,
Employer

And

Director, Office of Workers' Compensation Programs,
Party-In-Interest

DECISION AND ORDER AWARDING BENEFITS IN LIVING MINER'S AND SURVIVOR'S CLAIMS

This proceeding arises from a claim for benefits under the Black Lung Benefits Act of 1977, 30 U.S.C. Section 901 et seq. In accordance with the Act and the regulations issued thereunder, the case was referred by the Director, Office of Workers' Compensation Programs for a formal hearing.

Benefits under the Act are awardable to miners who are totally disabled within the meaning of the Act due to pneumoconiosis, or to the survivors of miners who were totally disabled at the time of their deaths (for claims filed prior to January 1, 1982), or to the survivors of miners whose deaths were caused by pneumoconiosis. Pneumoconiosis is a dust disease of the lungs arising from coal mine employment and is commonly known as "black lung."

A formal hearing was held before the undersigned on November 8, 2006, in Charleston, West Virginia, at which all parties were afforded full opportunity in accordance with the Rules of

Practice and Procedure (29 C.F.R. Part 18) to present evidence and argument as provided in the Act and the regulations issued thereunder, set forth in Title 20, Code of Federal Regulations, Parts 410, 718, 725, and 727. At the hearing, I admitted Director's Exhibits (SDX) 1 through 28, Employer's Exhibits (SROX) 2 through 4, 6, 8, and 11 through 21, and Claimant's Exhibits (SCX) 1 through 10. The Claimant was provided with additional time to submit exhibits, and on January 25, 2007 I admitted Claimant's Exhibits 1 (amended), 4, and 6 into the record in connection with the survivor's claim.

In connection with the living miner's claim, I admitted Director's Exhibits (LMDX) 1 through 61; Claimant's Exhibits (LMCX) 1 through 10; and Employer's Exhibits (LMROX) 2, 4 and 5, 8, 12, and 16 through 21. The Claimant was provided with additional time to submit exhibits, and on January 25, 2007 I admitted Claimant's Exhibits 1 (amended), 8, and 10 into the record in connection with the living miner's claim.

The parties were provided with time to file post-hearing briefs. Claimant filed her briefs on February 27, 2007; the Employer filed its brief on March 1, 2007; the Director did not file a brief.

I have based my analysis on the entire record, including the exhibits, submitted briefs, and representations of the parties, and given consideration to the applicable statutory provisions, regulations, and case law, and made the following findings of fact and conclusions of law.

Jurisdiction and Procedural History[1]

Mr. A. filed a claim for benefits on October 1, 2002 (LMDX 3). On January 20, 2004, the District Director (Director) issued a Proposed Decision and Order awarding benefits on Mr. A.'s claim (LMDX 32). The Employer appealed, and requested a hearing before an Administrative Law Judge.

Mr. A. filed a previous claim on June 4, 1991, which was denied by the Director on May 22, 1979 (DX 1). Mr. A. did not further pursue this claim.

The Claimant is Mr. A.'s wife; she filed a claim for benefits as his survivor on February 24, 2005 (SDX 2). The Director issued a Proposed Decision and Order awarding benefits on December 7, 2005 (SDX 21). The Employer appealed, and requested a hearing before the Office of Administrative Law Judges, and the claim was forwarded to the Office of Administrative Law Judges.

Issues

The issues contested by the Employer in connection with the living miner's claim are:

[1] Both of these claims fall under the evidentiary restrictions of the new regulations. While they were consolidated for hearing purposes, the evidentiary record in each claim has been kept separate. I have designated the evidence in the living miner's claim as "LMDX" for the Director's Exhibits; "LMCX" for the Claimant's Exhibits, and "LMEX" for the Employer's Exhibits. In connection with the survivor's claim, I have designated the evidence as "SDX" for the Director's Exhibits," "SCX" for the Claimant's Exhibits, and "ESX" for the Employer's Exhibits.

1. Whether Mrs. A. has established that Mr. A. had pneumoconiosis.
2. If so, whether Mr. A.'s pneumoconiosis was due to his coal mine employment.
3. Whether Mr. A. was totally disabled.
4. If so, whether Mr. A.'s totally disabling respiratory condition was due to pneumoconiosis.
5. Whether the evidence establishes a material change in condition.

In connection with the survivor's claim, in addition to the issue of whether Mr. A. had pneumoconiosis due to his coal mine employment, the Employer disputes that Mr. A.'s death was due to pneumoconiosis (Tr. 46-47).

Stipulations

The Employer agrees that Mr. A. had 19 years of coal mine employment, and that it is properly designated as the responsible operator. In addition, the Employer agrees that Mrs. A. was Mr. A.'s dependent, and that she qualifies as his survivor (Tr. 46-47).

Findings of Fact and Conclusions of Law

Background

Mr. S. L. A., the miner, was born on November 18, 1941. He married M. F. M., the Claimant, on December 24, 1980, and they remained married until Mr. A.'s death on January 14, 2005. Mr. A. began working in the coal mines when he was 18 years old, and he retired in 1989. Mr. A.'s Social Security Earnings records reflect that he worked for 28 years in coal mine employment; his jobs included buggy operator, bolt machine operator, hand loader, and shuttle car operator.

DISCUSSION[2]

Living Miner's Claim

In connection with Mr. A.'s living miner's claim, the parties submitted the following evidence, in accordance with the limitations of the new guidelines.

X-ray Evidence[3]

[2] I have relied on the evidence summary forms submitted by the parties, as amended by counsel during the hearing.

[3] B-B reader; and BCR - Board Certified Radiologist. These designations indicate qualifications a person may possess to interpret x-ray film. A "B Reader" has demonstrated proficiency in assessing and classifying chest x-ray

Exhibit No.	*Date of X-ray*	*Reading Date*	*Physician/ Qualifications*	*Impression*
LMEX 4	8-1-97	2-8-05	Wheeler/B, BCR	Negative for pneumoconiosis
LMXC 1	8-1-97	11-10-04	Alexander/B, BCR	2/2, q, p, category B opacities
LMEX 8	9-15-98	2-8-05	Scott/B, BCR	Negative for pneumoconiosis
LMCX 1	9-15-98	11-10-04	Alexander/B, BCR	2/2, q, p, category B opacities
LMDX 19	12-30-02	3-4-03	Binns/B, BCR	Read for quality purposes
LMDX 18	12-30-02	12-30-02	Patel/B, BCR	2/3, t, s; category A opacities
LMDX 29	12-30-02	8-19-03	Wheeler/B, BCR	Negative for pneumoconiosis
LMCX 1	12-30-02	11-10-04	Alexander/B, BCR	2/2, q, p, category B opacities

CT Scan Evidence

Mr. A. underwent a CT scan on February 26, 2003. Dr. Rose, the radiologist who reviewed this scan, noted innumerable tiny nodules scattered through the interstitium bilaterally, most consistent with pneumoconiosis. He also described larger densities in the lateral left upper zone, extending to the apex, and in the posterior right mid to upper zone, with some calcification. Dr. Rose reviewed Mr. A.'s x-rays dating back to 1998, concluding that the nodular densities were chronic, and likely reflected either progressive massive fibrosis associated with the pneumoconiosis, or superimposed granulomatous change. He was concerned by a 2.2 cm. focal mass in the left hilum, which he felt was suspicious for carcinoma, because it was disproportionately enlarged in relation to the other mediastinal lymph nodes. His impression was pneumoconiosis with superimposed progressive massive fibrosis or granulomatous change, and a new finding of a left hilar mass lesion that was suspicious for central bronchogenic carcinoma.

Dr. Alexander also reviewed this CT scan (LMDX 46). He noted innumerable tiny round densities bilaterally, consistent with a background of pneumoconiosis. He also noted bilateral peripheral pleural-based partially calcified large opacities consistent with progressive massive fibrosis of complicated pneumoconiosis. Dr. Alexander felt that the radiographic appearance of these masses was much more typical of pneumoconiotic conglomerate fibrosis than granulomatous disease, because in granulomatous disease, the borders of the masses would be

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evidence for pneumoconiosis by successful completion of an examination. A "Board Certified Radiologist" has been certified, after four years of study and an examination, as proficient in interpreting x-ray films of all kinds including images of the lungs.

smooth and regular, and there should be solitary calcified pulmonary nodules, of which there were none.

Dr. Wheeler reviewed the February 26, 2003 CT scan, concluding that it showed no pneumoconiosis (LMEX 8). He described probable healed conglomerate granulomatous disease, compatible with tuberculosis or histoplasmosis, with a mass in the posterior right upper lung and lower right apex, and lower posterior right upper lung, and a small mass in the left apex, all containing calcified granulomata and involving the pleura. There was a 3 cm. mass in the lower lateral left hilum, compatible with adenopathy or cancer, and small granulomata, more likely than tumors, involving the pleura in both lower lobes. There were also small calcified granuloma in the upper right hilum, and minimal emphysema, with areas of decreased upper lung markings. Dr. Wheeler indicated that the masses in Mr. A.'s lungs were not large opacities of coal workers' pneumoconiosis, because they were calcified, they were peripheral involving the pleura, and there were no symmetrical small background nodules in the central portion of the mid and upper lungs.

Mr. A. also underwent a PET scan on March 24, 2003. While the actual report is not in the record, Dr. Alexander reproduced the report by Dr. Robert Smith as an appendix to his January 10, 2005 report (LMCX 3). Dr. Smith reported that the clinical indication for the scan was an abnormal right lung mass, with bilateral hilar adenopathy, shown on the February 26, 2003 CT scan.[4] Dr. Smith noted a pathologic focus of hypermetabolic FDG uptake corresponding to the known posterior right upper lobe mass, highly suspicious for malignancy. He also noted pathologic foci of hypermetabolic FDG uptake corresponding to the right and left hilar lymph nodes, and the right paratracheal lymph node regions. According to Dr. Smith, the pathologic focus of uptake was particularly intense in the left hilar lymph node region. Dr. Smith also felt that these findings were highly suspicious for right upper lobe primary malignancy with metastatic disease to the hilar lymph nodes bilaterally.

Dr. Smith cautioned that Mr. A. had a history as a coal miner, and that occasionally coal workers' pneumoconiosis that presents with multiple nodular densities in the lung fields may cause hypermetabolic FDG uptake. He recommended a CT guided needle biopsy of the right upper lobe mass lesion.

Pulmonary Function Studies

Exhibit No.	***Date***	***Age/Ht***	***FEV1***	***FVC***	***MVV***	***Effort***	***Qualifying***[5]

[4] In fact the "abnormal" lung mass that precipitated the PET scan was in Mr. A.'s left lung.

[5] A "qualifying" pulmonary function study yields values that are equal to or less than the appropriate values set out in the tables at 20 C.F.R. Part 718, Appendix B. A "non-qualifying" study exceeds those values. 20 C.F.R. §718.204(b)(2)(i).

Exhibit No.	*Date*	*Age/Ht*	*FEV1*	*FVC*	*MVV*	*Effort*	*Qualifying*[5]
LMDX 17	12-30-02	60/72	2.90	5.32	116	Good	No
LMDX 30	9-24-03	61/72	2.94 2.97*	5.58 5.43*			No No

* After administration of bronchodilators

Arterial Blood Gas Studies

Exhibit No.	*Date*	*Physician*	*pCO2*	*pO2*	*At Rest* *After Exercise*
LMDX 12	12-30-02	Rasmussen	34 30	74 88	At Rest After Exercise
LMDX 30	9-24-03	Zaldivar	35 25	87 110	At Rest After Exercise

Medical Opinions

Dr. D. L. Rasmussen

Dr. Rasmussen examined Mr. A. on December 30, 2002 at the Director's request (LMDX 15). He reported Mr. A.'s history of coal mine employment, as well as his family, social, and medical history, and symptoms. Dr. Rasmussen reported that Mr. A. had smoked about a pack of cigarettes a day since 1956. On his examination of Mr. A., Dr. Rasmussen noted moderate to marked reduction of breath sounds on auscultation, and an increased expiratory phase with forced respirations. Dr. Rasmussen administered an x-ray, which showed pneumoconiosis t/s 2/3 in all lung zones, as well as a category A opacity. Mr. A.'s pulmonary function studies showed a minimal obstructive ventilatory impairment, and his arterial blood gas test results were normal.

Dr. Rasmussen concluded that Mr. A. had coal workers' pneumoconiosis, category A, based on his 33 year history of coal mine employment, and his x-ray findings; chronic bronchitis, based on his chronic productive cough; and possible heart disease, based on his abnormal ECG. He attributed Mr. A.'s pneumoconiosis to his exposure to coal mine dust, his chronic bronchitis to his exposure to coal mine dust and cigarette smoking, and his possible heart disease to non-occupational factors. Dr. Rasmussen stated that Mr. A. had poor exercise tolerance, with an

abnormal exercise ECG; he had minimal loss of lung function. Dr. Rasmussen felt that Mr. A. retained the pulmonary capacity to perform his last regular coal mine job. He had category A complicated pneumoconiosis, and he could have heart disease. According to Dr. Rasmussen, the two risk factors for Mr. A.'s minimal impairment were his cigarette smoking and his exposure to coal mine dust.

Dr. Steven L. McCormick

Dr. McCormick evaluated Mr. A. for consideration of cardiac catheterization on February 5, 2003, on referral by Dr. Robert Stanley (LMDX 21). He reported that Mr. A. had a longstanding history of fatigue and shortness of breath, and palpitations, and that he had severe chronic lung disease. A stress test performed the previous December showed significant ST segment depression. Dr. McCormick performed the catheterization, and reported that the study was normal, with a normal ventricular function. He stated that Mr. A. had no significant epicardial coronary artery disease. He also enclosed a copy of Mr. A.'s chest x-ray, which suggested a left perihilar mass that needed to be followed up.

In his history and physical report, Dr. McCormick noted that Mr. A. had a history of emphysema and chronic obstructive pulmonary disease, and that he was undergoing evaluation for pneumoconiosis. Mr. A. was short of breath with exertion. On his examination of Mr. A., Dr. McCormick noted that his chest sounds were decreased, with some rhonchi in the bases.

An x-ray performed at Dr. McCormick's request was read by Dr. John Willis, who reported a background of small parenchymal opacities consistent with a history of occupational pneumoconiosis. He also noted larger masses in the right upper lobe, and laterally in the left mid lung zone, as well as enlargement of the left hilum, particularly along its inferior aspect, which appeared to represent a circumscribed mass. He stated that while all of the findings could well represent pneumoconiosis, it was unusual to have a mass contiguous with the hilum as a conglomerate mass of pneumothorax (*sic*). He was concerned that this could represent a neoplasm, and recommended comparison with old x-rays, or a CT scan.

Dr. Robert C. Stanley

Dr. Stanley, who is with the Charleston Area Medical Center, was one of Mr. A.'s treating physicians, and his records are part of the exhibit file (LMDX 51). They include a report on an x-ray performed on April 3, 2003, after Mr. A. underwent thoracotomy. Dr. Dameron noted a small left pneumothorax, and vascular congestion, as well as a redemonstration of a right upper lobe mass. He indicated that Mr. A. was known to have coal workers' pneumoconiosis, and that there was tissue sampling of a right lung mass on March 31, 2003, after Mr. A. underwent CT guided biopsy. A chest x-ray performed on April 5, 2003 was read by Dr. Elkins to show no change in the bilateral pneumothoraces or pulmonary infiltrates.

A chest x-ray was performed on March 31, 2003, and read by Dr. Skeens, who noted a vague area of increased density in the left perihilar region for which neoplasm would be a consideration, and opacities in the right upper and left peripheral thorax, which he felt represented conglomerate masses.

Mr. A. underwent a chest CT scan on February 26, 2003. As discussed above, Dr. Colin Rose, who reviewed the films, noted moderately severe air trapping consistent with COPD, as well as innumerable tiny nodules scattered through the interstitium bilaterally, most consistent with pneumoconiosis. Dr. Rose described larger densities in the left upper zone laterally, extending to the apex, and in the right mid to upper zone posteriorly. He noted some calcification within these lesions. Dr. Rose reviewed previous x-rays back to 1998, and concluded that the nodular densities were chronic, and likely reflected either progressive massive fibrosis associated with the pneumoconiosis, or superimposed granulomatous change.

Dr. Rose described a 2.2 cm. focal mass in the left hilum, which could reflect further adenopathy, or bronchogenic carcinoma. Because it was disproportionately enlarged in relation to the other mediastinal lymph nodes, he felt that it should be considered fairly suspicious for a central bronchogenic carcinoma; he recommended bronchoscopy. Dr. Rose's impression was pneumoconiosis with superimposed progressive massive fibrosis or granulomatous change, and a new finding of a left hilar mass lesion that was fairly suspicious for central bronchogenic carcinoma.

Dr. Stanley's records include an x-ray report dated February 4, 2003, which was prepared by Dr. Willis.[6] He noted a background of small parenchymal opacities consistent with a history of occupational pneumoconiosis, and larger masses in the right upper lobe and laterally in the left mid lung zone. He also described enlargement of the left hilum, particularly along its inferior aspect, which appeared to represent a circumscribed mass. He felt that while all of the findings could represent pneumoconiosis, it was unusual to have a mass contiguous with the hilum as a conglomerate mass of pneumothorax (*sic*). He suggested comparison with previous x-rays and CT scan.

Dr. Stanley's records include a report of a September 15, 1998 x-ray that was read by Dr. Cruz, who indicated that the x-ray showed no significant change since an August 1997 study. He noted numerous small ill-defined opacities again seen throughout both lungs, with some patchy densities at the upper lung fields, consistent with pneumoconiosis complicated with bilateral pulmonary fibrosis.

Dr. John L. Chapman

[6] It appears that this is the same x-ray discussed in Dr. McCormick's records, which was dated February 5, 2003.

Dr. Chapman saw Mr. A. on March 11, 2003, on referral from Dr. Robert Stanley (LMDX 21). He reported that Mr. A. was a disabled coal miner with a long history of COPD/emphysema/black lung, and coal workers' pneumoconiosis. Dr. Chapman indicated that a CT scan showed bilateral changes consistent with fibrosis/pneumoconiosis. There was a focal mass in the left hilum that was not present on previous films, and that was worrisome in appearance. There was an additional rather large conglomerate mass at the right base. Dr. Chapman indicated that he would request fine needle aspiration of the right mass, along with a PET scan, bronchoscopy, and pulmonary function tests.

Dr. Chapman saw Mr. A. on March 31, 2003 for complaints of iatrogenic pneumothorax. He noted that Mr. A. had a history of known chronic obstructive pulmonary disease/coal workers' pneumoconiosis, and that he was undergoing surgical evaluation for bilateral lung masses. He had a history of a stable mass in the right lower lobe area, but there was a new left hilar mass that was fairly suspicious for a central bronchogenic carcinoma. Flexible bronchoscopy did not show evidence of an endobronchial lesion. On his examination of Mr. A., Dr. Chapman noted that his lungs were clear bilaterally. He admitted Mr. A. for chest tube placement to re-expand his right lung.

Dr. Chapman performed a bronchial washing of the left hilar mass lesion shown on CT scan on March 25, 2003, which was changing in appearance; he indicated that Mr. A. had known coal workers' pneumoconiosis with a stable right basilar mass, and a left hilar mass that was changing in appearance (LMDX 21). The pathology report showed no malignant cells. Dr. Chapman performed a needle aspiration of a 2.2 cm focal mass from Mr. A.'s right lung on March 31, 2003 (LMDX 21). The pathology report showed fibrosis and anthracotic pigment, but no evidence of malignancy. Dr. Chapman performed a biopsy of a mass from Mr. A.'s left lung on April 3, 2003 (LMDX 21). The pathology report subsequently showed a fragment of a poorly differentiated squamous cell carcinoma with necrosis and sclerosis.

Dr. Chapman prepared the discharge summary in connection with Mr. A.'s admission to the Charleston Area Medical Center on March 31, 2003, for a left thoracotomy (LMDX 21). He indicated that Mr. A. had known chronic obstructive pulmonary disease/coalworkers' pneumoconiosis, and was undergoing a surgical evaluation for bilateral lung masses. According to Dr. Chapman, Mr. A. had a history of a stable mass in the right lower lobe area. But there was a new left hilar mass, fairly suspicious for carcinoma. Dr. Chapman noted a 45 pack year history of cigarette smoking. The pathology report in connection with the thoracotomy showed poorly differentiated squamous cell carcinoma with necrosis and sclerosis.

An x-ray performed on March 31, 2003 was reviewed by Dr. Joseph Skeens, who noted a vague area of increased density in the left perihilar region, with neoplasm being a consideration; and opacities in the right upper thorax and left peripheral thorax that he favored as representing conglomerate masses (LMDX 21).

An x-ray was performed at Dr. Chapman's request on May 13, 2003 (LMDX 21). Dr. Robert Smith reviewed this x-ray, noting chronic appearing interstitial lung markings with fibronodular changes and vague densities in the upper lobes bilaterally, most consistent with occupational pneumoconiosis. There was also prominence of the right and left hilar shadows, and Dr. Smith could not exclude possible adenopathy and/or recurrent neoplastic disease, in a patient with a known clinical history of lung malignancy. He recommended further evaluation by CT scan.

Mr. A. underwent a left thoracotomy on May 19, 2003, with a preoperative diagnosis of bilateral lung masses (LMDX 21). Dr. Chapman reported that a biopsy was taken of the left lung mass. He noted further nodular areas in the left lung; the frozen section came back squamous cell carcinoma, poorly differentiated.

Dr. Richard L. Naeye

Dr. Naeye reviewed medical evidence at the Employer's request, and examined a tissue slide, and prepared a report dated January 29, 2004 (LMDX 31). The tissue slide was from the biopsy that was performed on April 7, 2003, and contained tissue from a lymph node. Dr. Naeye reported that there were no findings of coal workers' pneumoconiosis in the very limited tissue available for microscopic review. He indicated that there was x-ray evidence of its presence, but that if it were present, the pulmonary function and arterial blood gas test results indicated that it was not causing any disability.[7] According to Dr. Naeye, Mr. A. apparently had inoperable squamous cell carcinoma in both lungs, the presumed consequence of his heavy cigarette smoking since his teenage years. He stated that neither U.S. nor European coal miners have an increased frequency of carcinoma of the lung when cigarette smoking is taken into consideration.

Dr. Naeye prepared a supplemental report dated September 11, 2006, after reviewing a report by Dr. Alexander (LMEX 12). Dr. Naeye disagreed with Dr. Alexander's claim that x-ray findings can establish the diagnosis of complicated coal workers' pneumoconiosis. He stated that complicated pneumoconiosis is not just a severe extension of simple pneumoconiosis; it is a unique disorder with immunologic histologic features that arises unexpectedly and expands rapidly in lungs where pneumoconiosis is already present. According to Dr. Naeye, it is the immunologic features that explain why it grows so rapidly, whereas the toxic products of silica are mainly responsible for the much slower growth of lesions of simple pneumoconiosis. He felt that none of these findings were shown in Mr. A.'s lungs. According to Dr. Naeye, complicated pneumoconiosis is not a diagnosis that can be made by x-ray without confirmation by direct tissue examination.

[7] It is unclear what medical records, if any, that Dr. Naeye had access to, as he did not indicate that he had reviewed any specific medical records.

Dr. Naeye prepared a supplemental report dated October 6, 2006, after examining two slides provided by the Employer (LMDX 13). The first slide apparently came from Mr. A.'s right lung, although there is no indication of the date that it was obtained, or the procedure from which it was taken.[8] According to Dr. Naeye, the slide contained smeared exfoliated cells, and a diagnosis of coal workers' pneumoconiosis cannot be made from an examination of exfoliated cells.

The second slide appears to have come from the March 31, 2003 core biopsy of Mr. A.'s right lung, although again the date is not indicated. According to Dr. Naeye, a diagnosis of coal workers' pneumoconiosis depends on a microscopic examination of intact lung tissues, in which the relationship between cellular structures can be identified. He indicated that the second slide had a 12 mm long core of tissue presumably removed from a lung by needle biopsy. According to Dr. Naeye, less than 1% of the lung tissue on this slide was occupied by black pigment, which was not accompanied by more than very rare birefringent crystals, tiny enough to have been fibrogenic. There was no fibrosis specifically associated with the crystals. He concluded that there were no lesions of coal workers' pneumoconiosis in the tissues, and almost no tiny birefringent crystals of toxic, fibrogenic silica, and no resulting fibrosis.

Dr. George L. Zaldivar

Dr. Zaldivar examined Mr. A. on September 24, 2003 at the Employer's request (LMDX 30). He reported Mr. A.'s employment history, as well as his family, social, and medical histories, and his symptoms. On his examination of Mr. A., Dr. Zaldivar noted that his lungs were clear to auscultation, with no wheezes, crackles, or rales. Dr. Zaldivar also administered an x-ray, as well as pulmonary function and arterial blood gas studies.[9] On the report of the pulmonary function studies, Dr. Zaldivar indicated that the results showed a moderate irreversible obstruction, hyperinflation with air trapping, and normal diffusion. Dr. Zaldivar's impression was history of inoperable left lung cancer, history of shortness of breath, no abnormal breath sounds, weight loss, likely as a result of lung cancer, longstanding smoking history, and history of work in the coal mines.

Dr. Zaldivar also reviewed medical records provided to him by the Employer. Dr. Zaldivar's findings included a normal cardiopulmonary stress test, a very high carbon monoxide level of a smoker of two packs of cigarettes a day, radiological evidence of cancer in the right lung and a background of simple pneumoconiosis, moderate irreversible airway obstruction, airtrapping by lung volumes with hyperinflation, and normal diffusion capacity. He indicated that Mr. A.'s pulmonary function study showed a moderate airway obstruction, which had no clinical significance given the normal results of the cardiopulmonary stress test. According to Dr. Zaldivar, Mr. A. had simple pneumoconiosis and cancer in his right lung, which had the

[8] Mr. A. underwent bronchoscopy with washings of the left hilar mass by Dr. Chapman on March 25, 2003; needle aspiration of a mass from his right lung on March 31, 2003, and a biopsy of his left lung mass on April 3, 2003.
[9] If Dr. Zaldivar read this x-ray, his report is not in the record. Instead, attached to Dr. Zaldivar's report is an interpretation of this x-ray done on October 22, 2003 by Dr. Wheeler.

radiographic appearance of complicated coal workers' pneumoconiosis. However, Mr. A.'s history clearly showed that the mass was not due to coal workers' pneumoconiosis, but was due to cancer that had spread, according to the PET scan, to both the right and left hilar areas and the right paratracheal lymph nodes.[10] Dr. Zaldivar felt that strictly from a pulmonary standpoint, Mr. A. was fully capable of performing his usual coal mining work or work of similar exertion.

Dr. Michael S. Alexander

Dr. Alexander reviewed Mr. A.'s medical records at the Claimant's request, and prepared a report dated November 10, 2004 (LMDX 46). He described the series of x-rays he had read, stating that it was very important to perform a "series reading" of the x-rays, and that to do so, he placed all of the x-rays on a light box in chronological order, allowing him to simultaneously view a time span of six years, and thus make an accurate determination of which abnormalities were chronic and stable, and which were new or acute. According to Dr. Alexander, all of the x-rays that he reviewed showed a background of small round opacities in all six lung zones, consistent with pneumoconiosis, category q/p, 2/2, with areas of coalescence in the upper zones. They were stable and unchanged. In addition, all of the x-rays showed large opacities consistent with complicated pneumoconiosis, located in both upper zones and both mid zones, more extensive on the right. They were also stable and unchanged. Both hilar regions were prominent, probably due to some degree of pulmonary arterial hypertension associated with chronic obstructive pulmonary disease; they were stable and unchanged.

Dr. Alexander also described a left infrahilar mass approximately 25 mm in diameter on the x-ray dated December 30, 2002, which was a new abnormal finding, and was again seen on subsequent 2003 x-rays. Based on the left thoracotomy performed on April 3, 2003, this was a poorly differentiated squamous cell lung cancer. There were no other new or acute findings, other than a right pneumothorax that occurred during the March 31, 2003 biopsy.

Dr. Alexander reviewed the February 26, 2003 chest CT scan. As discussed above, he noted innumerable tiny round densities bilaterally on the lung parenchymal settings, consistent with a background of pneumoconiosis. There were also bilateral peripheral pleural-based partially calcified large opacities consistent with progressive massive fibrosis of complicated pneumoconiosis. According to Dr. Alexander, the radiographic appearance of these masses was much more typical of pneumoconiotic conglomerate fibrosis than granulomatous disease, because in granulomatous disease, the borders of the masses would be smooth and regular, and there should be solitary calcified pulmonary nodules, of which there were none. He noted that the other radiologists who reviewed x-rays and the CT scan concurred in the diagnosis of conglomerate masses of complicated pneumoconiosis.

[10] Dr. Zaldivar reported that the March 24, 2003 PET scan was read as showing hypermetabolic uptake of the partially calcified right upper lobe nodules, and the right paratracheal area and both hilar areas were suspicious for lymph node metastasis.

But most importantly, according to Dr. Alexander, the March 31, 2003 needle biopsy of the right upper lobe mass showed "Lung with fibrosis and anthracotic pigment. No evidence of malignancy." He stated that the presence of carbon pigment in this mass indicated the inhalation of coal dust, and thus pneumoconiosis as the cause of the mass. The right upper zone mass measured approximately 60 mm in greatest dimension, the left upper zone mass measured 30 mm, the right mid zone mass measured approximately 10 mm., and the left mid zone mass measured 20 mm. As the summed diameter of the masses was greater than 50 mm., they constituted category B complicated pneumoconiosis. Dr. Alexander found no pleural abnormalities that were characteristic of asbestos exposure, and no pleural effusion, significant emphysematous changes, or bronchiectasis.

With respect to the left lung, Dr. Alexander noted an abnormal round soft tissue mass in the lower left hilar region, about 20 mm. in diameter, which corresponded in size and location to the abnormality seen on the December 30, 2002 and subsequent x-rays, and in the location of the focal area of hypermetabolic activity seen on the March 24, 2003 PET scan. The mass was subsequently determined to be a poorly differentiated squamous cell cancer of the lung during a thoracotomy and open lung biopsy performed on April 3, 2003.

Dr. Alexander reviewed the PET scan performed on March 24, 2003, noting an area of mild to moderate uptake in the posterior portion of the right upper lung, corresponding in location to the dominant large opacity seen on chest x-rays and CT scan. There was a smaller, less defined area of mild increased uptake posteriorly in the left upper zone, and a few tiny round areas of slightly increased uptake in the mediastinum, probably corresponding to reactive (non-malignant) lymph nodes. Most notable was a focal round area of significantly increased uptake in the left hilum, which corresponded in size and location to the mass seen on x-rays and CT scan that was subsequently proven to be a poorly differentiated squamous cell cancer by open thoracotomy biopsy. According to Dr. Alexander, the degree of uptake in the left hilar mass was nearly 100% greater than the uptake seen in the right upper lobe mass, which was essentially the same as the mediastinal lymph nodes.

According to Dr. Alexander, the appearance of progressive massive fibrosis in complicated pneumoconiosis on PET scan has been described as occasionally showing amorphous areas of slightly increased uptake, thought to represent an ongoing fibrotic response in the large masses of complicated pneumoconiosis. Masses in which the deposition of fibrin and collagen have ceased, or masses that are predominantly calcified would not be expected to show uptake. He felt that the appearance and degree of uptake seen in the posterior right upper zone was most consistent with progressive massive fibrosis of complicated pneumoconiosis, and should not be interpreted as suspicious for a lung cancer. In contrast, the intense focal activity seen in the left hilum was very characteristic of a lung malignancy.

Dr. Alexander discussed other studies, including a pulmonary function study on March 24, 2003, which showed mild to moderate abnormalities, findings that were nonspecific and could be seen in pneumoconiosis. He stated that the bronchial washings obtained during the

March 25, 2003 flexible bronchoscopy did not show the presence of malignant cells or any endobronchial lesion. He felt this was noncontributory, as a more definitive diagnostic test was performed later. A fine needle aspiration biopsy of the peripheral right upper lung mass done on March 31, 2003 showed a lung with fibrosis and anthracotic pigment, and no evidence of malignancy, and was diagnostic of complicated pneumoconiosis. Finally, the April 3, 2003 left thoracotomy biopsy results on the left intrahilar mass showed the presence of poorly differentiated squamous cell carcinoma. According to Dr. Alexander, this diagnosis of malignancy in the left lung was independent of the diagnosis of complicated pneumoconiosis.

Based on his review of the medical evidence, Dr. Alexander concluded, beyond a reasonable degree of medical certainty, that Mr. A. suffered from two simultaneous disease processes, independent of each other. He had complicated pneumoconiosis category B, with bilateral conglomerate masses of progressive massive fibrosis; at some time between 1998 and 2002, he also developed a lung cancer adjacent to the left hilum.

Dr. Alexander prepared a supplemental report dated December 21, 2004, after reviewing reports by Dr. Zaldivar, Dr. Naeye, and Dr. Castle (LMDX 53).[11] As he stated in his earlier report, it was his opinion that Mr. A. suffered from complicated coal workers' pneumoconiosis as well as lung cancer. These physicians thought that Mr. A. had only simple pneumoconiosis in addition to lung cancer. To put his opinions in perspective, Dr. Alexander set out a time line of clinic visits and diagnostic tests.

Mr. A. saw Dr. Rasmussen in connection with his black lung claim in December 2002. Dr. Rasmussen found that he had coal workers' pneumoconiosis, and an abnormal EKG stress test. Mr. A. then visited Dr. McCormick on February 4, 2003, in connection with his abnormal EKG and stress test. Dr. Willis, the radiologist who read an x-ray requested by Dr. McCormick, reported that the larger masses in Mr. A.'s lungs were due to conglomerate masses of complicated pneumoconiosis, and that there was a mass in the left perihilar region atypical for pneumoconiosis; he recommended further evaluation.

Mr. A. then had a CT scan performed on February 26, 2003. Dr. Alexander noted that the interpreting radiologist, Dr. Rose, reviewed x-rays dating back to 1998, a retrospective analysis that Dr. Alexander felt was critical to establish which abnormalities were chronic and stable, versus abnormalities that were new and acute. He pointed out that neither Dr. Zaldivar nor Dr. Naeye performed such a comprehensive radiographic review. Thus, he felt that he and Dr. Rose had a more comprehensive vantage point from which to make a more accurate diagnosis. Both Dr. Alexander and Dr. Rose concluded that the larger densities or masses in Mr. A.'s lungs were chronic, and most likely represented progressive massive fibrosis of complicated pneumoconiosis. There was a new finding of a 2.2 cm. focal mass in the left hilum, suspicious for a central bronchogenic carcinoma.

[11] Dr. Castle's report is not part of the record in the living miner's claim.

Both Dr. Alexander and Dr. Rose felt that the large pulmonary opacities and the left hilar mass were NOT related, which was clearly shown by the chronological survey. They felt that the left hilar mass represented a primary lung cancer, not metastatic adenopathy, and that the large pulmonary opacities were benign, and not representative of lung cancer. He stated that these points were critical, especially in light of the significant difference of opinion between the report on the PET scan and the interpretation of the CT scan. In his opinion, the PET scan was incorrectly interpreted by Dr. Smith, and his incorrect diagnosis was cited as "documented evidence" by Dr. Zaldivar and Dr. Naeye.

Mr. A. underwent a PET scan on March 24, 2003. Dr. Alexander disputed several portions of the report. He noted that the statement of the "clinical indication" was "just wrong:" Dr. Smith indicated that the study was for an abnormal right upper lobe mass with bilateral hilar adenopathy as demonstrated on the CT scan of February 26, 2003. But according to Dr. Alexander, that CT scan did not indicate bilateral hilar adenopathy, or suspicion of malignancy concerning the right upper lobe mass, since bilateral chronic large opacities were present. It did indicate an abnormal 2 cm left hilar mass that was very suspicious for a primary bronchogenic carcinoma. Dr. Smith did not realize that the intense activity in the left hilar region on the PET scan corresponded to the main abnormality seen on the CT scan, and represented a lung cancer. Erroneously, according to Dr. Alexander, he equated the less intense degree of uptake in the right hilum and paratracheal region to metastatic adenopathy. He also incorrectly attributed a much greater degree of suspicion for malignancy to the right upper lobe mass than it deserved. Dr. Alexander stated that the uptake in the right upper lobe mass was much less intense than the uptake in the left hilar mass, and therefore much less suspicious for malignancy. He stated that such a degree of mild uptake can be seen in conglomerate masses of complicated pneumoconiosis.

According to Dr. Alexander, this misreading of the PET scan was then incorporated into the other physicians' reports, and their references to this report should be discounted as misleading. This PET scan report indicated that the right upper lobe mass was the primary lung cancer, and that there was right paratracheal and bilateral hilar adenopathy. But the correct radiographic diagnosis, incorporating all of the radiographic information back to 1997, and a proper interpretation of the PET scan, is that the mass in the right upper zone was benign and due to complicated pneumoconiosis, not malignancy; and the area that was highly suspicious for malignancy was in the left hilum; there was no conclusive evidence of metastatic adenopathy. Dr. Alexander stated that these conclusions were borne out by the subsequent tissue biopsies and pathology reports.

Dr. Alexander noted that on March 25, 2003, Mr. A. underwent flexible bronchoscopy with bronchial washings; no malignant cells were found. Mr. A. then underwent needle biopsy of the right upper lobe mass, based on Dr. Smith's interpretation of the PET scan, on March 31, 2003. Dr. Alexander felt, contrary to some of the clinical notes, that the lung tissue sample was not inadequate or insufficient for diagnosis. He pointed to the clear statement in the cytology aspiration report by Dr. Riefsteck that the biopsy showed no evidence of malignancy, and that the right upper lobe mass showed "lung with fibrosis and anthracotic pigment." Dr. Alexander

stated: "What more histological proof is necessary to establish the diagnosis of a chronic coal-dust induced mass?" He felt that the other physicians either ignored this report, or did not attribute to it the diagnostic significance it deserved.

On April 3, 2003, Mr. A. underwent a left open thoracotomy to assess his left hilar mass. Dr. Alexander felt that the preoperative diagnosis of "bilateral lung nodules" was misleading, as they actually represented the stable large opacities of complicated pneumoconiosis, and the actual indication for this invasive surgical procedure was to evaluate the new suspicious left hilar mass. It established two crucial findings. First, the left hilar mass was caused by a non-small cell carcinoma, supporting the assessments of Dr. Alexander and Dr. Rose, and refuting that of Dr. Smith. Second, there were further nodular areas on the left lung, indicating metastatic disease, but not in the location or to the extent stated in the PET scan report.

Having set out this timeline, Dr. Alexander addressed the other reports. With respect to Dr. Zaldivar's report, Dr. Alexander noted that Dr. Wheeler acknowledged the presence of bilateral large opacities greater than one centimeter in size, and postulated that they were due to tuberculosis. However, Dr. Zaldivar indicated that Mr. A. had no history of tuberculosis. According to Dr. Alexander, if tuberculosis were the cause of the large pulmonary masses, it certainly would have been evident clinically, and documented in Mr. A.'s medical history. He felt that tuberculosis could be excluded as the cause of the large pulmonary opacities.

Dr. Alexander felt that Dr. Zaldivar's report indicated that he was aware that the biopsy of the right upper lobe mass contained fibrosis and anthracotic pigment, with no evidence of malignancy. But he did not understand why Dr. Zaldivar ignored those pertinent facts in his Findings and Comments. Dr. Alexander stated that Dr. Zaldivar's claim that there was radiological evidence of cancer in the right lung had absolutely no basis in fact, and that it was wrong for him to present this as an established hospital record. He also felt that one had to completely discount Dr. Zaldivar's comment that Mr. A. had a "cancer in the right lung, which has the appearance, radiographically, of complicated Coal Worker's Pneumoconiosis." In fact, according to Dr. Alexander, Mr. A. had complicated coal workers' pneumoconiosis that looked just like complicated coal workers' pneumoconiosis radiographically. Dr. Alexander stated:

> The rest of his statement in D8 is simply backwards, erroneous and misleading, verging on malpractice. In rebuttal, clearly the mass in the right lung **is due to** Coal Worker's Pneumoconiosis and **not due to** cancer, and there is no PET scan evidence that the primary lung cancer, which is actually in the **left hilum**, has spread to contralateral lymph nodes. In my opinion, Dr. Zaldivar's comments and opinions should be regarded as invalid since they do not acknowledge the presence of complicated Coal Worker's Pneumoconiosis and they mislead the reader into thinking that Mr. [A] has extensive metastatic lung cancer." (emphasis in original)

Dr. Alexander again discussed Dr. Wheeler's x-ray reading, characterizing his statement that the masses were not due to pneumoconiosis because the profusion of nodules was minimal, and the masses were peripheral with probable pleural involvement, as "fairly absurd." According to Dr. Alexander, it is a well known fact that as simple pneumoconiosis progresses to complicated pneumoconiosis with the development of large conglomerate masses, the apparent profusion of small opacities actually decreases, as they are incorporated into the larger masses, and also because the lung parenchyma becomes attenuated by emphysema and thoracic distortion. Dr. Alexander stated that large masses of complicated pneumoconiosis can be centrally or peripherally located, or both, and occasionally can extend to the pleural surface causing pleural involvement.

Finally, Dr. Alexander addressed Dr. Naeye's report, noting that his main contribution was his review of one glass slide from the April 3, 2003 thoracotomy. This biopsy was from the left hilar mass that was suspicious for cancer, and it therefore made sense that the lymph node was largely replaced by poorly differentiated squamous cell carcinoma. Dr. Alexander felt that because there was no suspicion by any radiologist that the left hilar mass was caused by pneumoconiosis, Dr. Naeye's statement that there was no black pigment and no birefringent crystals of silica in the tissues was totally unnecessary and actually misleading. Dr. Alexander indicated that one would not expect to find pneumoconiosis in this lymph node biopsy, but more importantly, this statement was then erroneously interpreted by other doctors to mean that there was no evidence of pneumoconiosis anywhere in Mr. A., when in fact the CT guided needle biopsy of the right lung mass done on March 31, 2003 was strongly diagnostic of pneumoconiosis.

Dr. Alexander pointed to Dr. Naeye's statement that in August of 2003 a chest x-ray showed lesions in both lungs, and that a biopsy performed on April 7, 2003 removed tissue that showed poorly differentiated squamous cell carcinoma, stating that it erroneously implied that the squamous cell carcinoma was found in the lesions of both lungs, whereas it was only found in the biopsy of the left hilar mass. He also felt that Dr. Naeye's statement that the widespread nature of the lesions on x-rays indicated that it was inoperable was wrong, because it was not the widespread nature of the lesions that indicated the lung cancer was inoperable, it was the fact that there were further nodular areas on the left lung, as noted in Dr. Chapman's April 3, 2003 operative report, that made the lung cancer inoperable. Dr. Alexander pointed out that Dr. Naeye acknowledged the presence of pneumoconiosis on x-rays, and felt that he should have realized that the chronic large lesions in both lungs represented conglomerate masses of complicated pneumoconiosis. But he felt that the larger problem was that when other doctors read this report, they took it to mean that there were no findings of pneumoconiosis anywhere in Mr. A.'s lungs.

Dr. Alexander stated that as a result of his occupational history, Mr. A. developed simple pneumoconiosis that progressed to complicated pneumoconiosis by 1997, and he then developed a primary lung cancer in his left hilar region, which was detectable on x-rays in 2002.

Dr. Alexander prepared a supplemental report dated November 30, 2006, after reviewing letters by Dr. Naeye dated September 11, 2006 and October 6, 2006 (LMCX 10). Dr. Alexander stated that although he had training and experience in pathology, it was not to the degree of

expertise that Dr. Naeye had; thus, he did not challenge Dr. Naeye's findings concerning the pathology in this case. According to Dr. Alexander, his only concern was that a large enough tissue sample was not obtained in order to confidently exclude the diagnosis of complicated pneumoconiosis. He noted that in his October 6, 2006 letter, Dr. Naeye indicated that the only tissue sample he had for evaluation was a 12 mm long core of tissue obtained by needle biopsy, which appeared to Dr. Alexander to represent a very limited sampling of a large conglomerate fibrotic mass.

Dr. Alexander agreed with Dr. Naeye that radiology is not as precise a medical science as pathology, noting that there was no better proof than an autopsy. But he stated that the science of radiology had been established for over one hundred years, and that the world's physicians rely on radiology to make reliable and precise diagnoses. He felt that Dr. Naeye was proposing that radiology be abolished as a diagnostic tool in establishing the diagnosis of complicated pneumoconiosis, which he thought was absurd. According to Dr. Alexander, he could cite to "innumerable" analogies where radiographic findings are relied on to indicate specific pathology, without the need for corresponding proof in the form of direct tissue examination. As an example, he noted that the combination of an enlarged heart, engorged pulmonary vasculature, and pleural effusions are used to make a confident diagnosis of congestive heart failure, without the need to insert a central venous pressure catheter to confirm the diagnosis. Rather, with the appropriate clinical history and abnormal findings on physical examination, the diagnosis of congestive heart failure can be established. He stated that, in the medical vernacular, certain time-established x-ray findings are termed pathognomonic, or distinctly characteristic of a particular disease or condition.

Dr. Alexander stated that this principle applies to the use of x-rays in pneumoconiosis: with the appropriate history of occupational exposure to coal dust, and the physical findings of pulmonary impairment, certain patterns and findings on x-rays are pathognomonic for both simple and complicated pneumoconiosis. In that small percentage of atypical or perplexing cases, there are now adjunct imaging studies, such as CT scan and PET scan, which were not clinically available in 1980 when the ILO standards were revised. According to Dr. Alexander, the diagnostic confidence of x-rays, CT scans, and PET scans is so reliable that tissue samples are rarely needed before definitive diagnosis is made, or surgery is undertaken. The one exception is oncology, where direct tissue examination of tumors is helpful in selecting the best course for cancer treatment.

Dr. Alexander discussed the establishment of standard radiographs by the ILO, and their use in evaluating pneumoconiosis. He noted that the ILO standard films are the gold standard of imaging for determining the presence and classification of pneumoconiosis. He also discussed the "Study Syllabus for the Classification of Radiographs of Pneumoconioses" produced by the Division of Respiratory Disease Studies, NIOSH, which is used by B readers to prepare for proficiency recertification. He noted that it does not once mention that complicated pneumoconiosis is a diagnosis that cannot be made without direct tissue examination, or that every patient suspected of having complicated pneumoconiosis on x-ray needs an invasive procedure or autopsy to prove it. He stated: "Unless Dr. Naeye has significant evidence to the contrary, the vast majority of B-readers are confident that they can make a diagnosis of

complicated Coal Worker's Pneumoconiosis that would stand up to pathologic scrutiny if necessary."

In the event that Dr. Naeye was suggesting that the rules for establishing a diagnosis of complicated pneumoconiosis needed to be changed, Dr. Alexander felt that there would be much resistance. He stated that it was simply impractical, expensive, and sometimes not possible to obtain tissue confirmation in every patient. He agreed that a properly performed autopsy would be definitive; but while a pulmonary impaired miner is alive, he did not think that it was necessary to subject him to a potentially problematic invasive surgical procedure.

Dr. Alexander stated that there have been decades of experience with plain x-ray findings which are pathognomonic for complicated pneumoconiosis. In difficult cases, this evaluation can be augmented with CT or PET scan. But there are radiographic x-ray standards that have been accepted since 1980 without requiring revision, and qualified pneumoconiosis B-readers who must pass a vigorous visual proficiency examination every four years. He stated: "It is obvious that x-ray findings can be relied on to establish the diagnosis of complicated Coal Worker's Pneumoconiosis. It is preposterous to claim that 'they cannot.'"

Dr. Steven M. Koenig

Dr. Koenig reviewed Mr. A.'s medical records at the Claimant's request, and prepared a report dated June 10, 2005 (LMCX 6). He summarized Mr. A.'s occupational and medical history, and noted that he smoked 1-2 packs of cigarettes a day for about 45 years.

Dr. Koenig concluded that Mr. A.'s exposure to coal mine dust was sufficient to cause respiratory impairment in a susceptible individual. He listed a number of indisputable objective findings, including evidence of obstructive lung disease, mildly decreased post-bronchodilator FEV1 with no significant improvement with bronchodilator, persistently decreased FEV1/FVC and FEV1 after bronchodilator, normal diffusing capacity, increased residual volume consistent with air trapping, increased RV/TLC, no evidence of restriction, evidence of moderately impaired exercise capacity, evidence of bilateral upper lobe large opacities on chest x-ray and CT scan, and non-small cell lung carcinoma in the left hilar region.

Dr. Koenig observed that the majority of physicians, including Dr. Zaldivar, agreed that Mr. A. had radiographic evidence of simple pneumoconiosis, and several bilateral large upper lobe opacities. All of the physicians agreed that the cause of the left hilar mass/opacity was non-small cell lung cancer, most consistent with poorly differentiated squamous cell carcinoma. But it was the cause of the other large opacities that was disputed. After his review of the medical records, Dr. Koenig favored complicated coal workers' pneumoconiosis as the cause of Mr. A.'s other large upper lobe opacities. In other words, he felt that Mr. A. developed lung cancer, which was the cause of his left hilar mass/large opacity, on a background of complicated pneumoconiosis.

According to Dr. Koenig, clearly the causes of upper lobe masses or large opacities on x-ray include lung cancer, chronic infections such as tuberculosis, and complicated pneumoconiosis. But he stated that when lung cancer and tuberculosis are the etiology, the

changes in the large opacities occur over a relatively short period of time. Citing to medical studies, Dr. Koenig stated that one of the best criteria for a lung opacity not being cancer is that there is no change over 18 to 24 months. Additionally, one of the criteria for differentiating complicated pneumoconiosis from tuberculosis is that tuberculosis is associated with rapid radiological changes. But the large opacities of complicated pneumoconiosis typically enlarge or progress much more slowly, over years, not months.

Thus, the fact that Mr. A.'s large upper lobe opacities, other than the left hilar mass, changed little over more than six years indicated to Dr. Koenig that complicated pneumoconiosis was the most likely cause of these opacities. He stated that if either lung cancer or tuberculosis were the etiology of the other opacities, and no treatment was given, significant progression and enlargement of the opacities would have occurred, likely with cavitation if they were due to tuberculosis. He pointed out that it was the rapid appearance of the new left hilar mass/large opacity that alerted Dr. Willis to the possibility that another etiology such as lung cancer could be at work.

Dr. Koenig also pointed to the results of the transthoracic CT guided lung biopsy that was performed on March 31, 2003. He stated that if cancer is the cause of a lung mass, particularly a very large one, the likelihood of such a biopsy finding cancer is very high. Thus, the negative biopsy mitigated very strongly against this right upper lobe lung mass being cancer. Additionally, the findings of lung fibrosis and anthracotic pigment were exactly what one would expect if one biopsied complicated pneumoconiosis. Nor was there any evidence of a granulomatous infection such as tuberculosis.

Dr. Koenig felt that the absence of systemic symptoms, such as fever, chills, sweats, loss of appetite, and weight loss before the appearance of the new left hilar lung cancer also indicated that complicated pneumoconiosis was the cause of the other large upper lobe opacities, and mitigated strongly against lung cancer and tuberculosis as the etiology. He noted that before the new left hilar mass/large opacity, no examiner reported that Mr. A. had even one of these symptoms. According to Dr. Koenig, tuberculosis is a chronic infection, and it is typically associated with fever, weight loss, and other systemic symptoms. Lung cancer is also associated with loss of appetite and weight loss. However, complicated pneumoconiosis is associated with primarily respiratory, but rarely systemic, symptoms. Both Dr. Zaldivar and Dr. Rasmussen reported that Mr. A. did not have a personal or family history of tuberculosis.

Dr. Koenig concluded that complicated coal workers' pneumoconiosis was by far the most likely cause of the other large opacities, or at the least, the right upper lobe opacity. He relied on the minimal change in Mr. A.'s large upper lobe opacities, other than the left hilar mass/large opacity over time, the absence of systemic symptoms such as fever, loss of appetite, fatigue, and weight loss before the development of the new left hilar mass, the absence of a history of or exposure to tuberculosis, and the CT guided biopsy of the right upper lobe mass/large opacity, which showed findings consistent with complicated pneumoconiosis and no evidence of malignancy or tuberculosis. He felt that the diagnoses of tuberculosis and lung cancer were untenable.

Dr. Koenig addressed Dr. Smith's interpretation of Mr. A.'s PET scan, noting that Dr. Smith favored a diagnosis of right upper lobe malignancy with metastasis to the right paratracheal and bilateral hilar lymph nodes. But Dr. Koenig pointed out that Dr. Smith recommended caution, and qualified his conclusion by stating that large opacities of complicated pneumoconiosis may occasionally cause hypermetabolic FDG uptake; indeed, because of this possibility, Dr. Smith recommended a CT guided biopsy of the right upper lobe mass to resolve the issue. This indicated to Dr. Koenig that Dr. Smith understood that hypermetabolic FDG uptake on a PET scan can be caused by metabolically active processes other than malignancy, including large opacities of complicated pneumoconiosis. Additionally, such uptake is not diagnostic of any abnormality, and a biopsy is always required to make a definitive diagnosis. Dr. Koenig noted that Dr. Smith indicated that the hypermetabolic FDG uptake was particularly intense in the left hilar lymph node region.

According to Dr. Koenig, when the PET scan findings were combined with the previous x-rays, which showed that the right upper lobe abnormality was stable since 1997, the new area of abnormality on x-ray, which was a left hilar mass in the area of the particularly intense FDG uptake, and which later proved to be non-small cell lung cancer, and the CT guided biopsy of the very large right upper lobe mass, which showed findings consistent with complicated pneumoconiosis but not malignancy, the only logical conclusion was that Mr. A. had a left hilar lung cancer on a background of complicated pneumoconiosis. Dr. Koenig stated that the cause of the particularly hypermetabolic left hilum on the PET scan was Mr. A.'s lung cancer, and the less hypermetabolic right upper lobe mass was due to complicated pneumoconiosis.

Dr. Koenig stated that Dr. Wheeler's opinions were not supported, and were in fact refuted by the medical literature. He noted that Dr. Wheeler attributed the masses/large opacities on x-ray to tuberculosis, because the profusion of nodules was minimal, and the masses were peripheral, probably involving the pleura. But according to Dr. Koenig, the medical literature establishes that complicated pneumoconiosis occurs most commonly in the apical posterior portions of the upper lobes, or the superior segments of the lower lobes, and starts as a mass near the periphery of the lung. In addition, as simple pneumoconiosis progresses to complicated, it is very common to see the apparent profusion of small opacities decrease, as a result of the small opacities being incorporated into the large opacities, and the development of emphysema and thoracic distortion.

Dr. Koenig noted that Dr. Binns attributed the right upper lobe abnormalities to either pneumonia or pneumoconiosis. Dr. Koenig felt that if Dr. Binns had access to the clinical information, which contained no evidence for an old infectious process, and the old x-rays, which showed a stable, chronic appearance to the right upper lobe abnormalities, he would have favored pneumoconiosis as the better diagnosis.[12]

Dr. Koenig pointed out that Dr. Naeye reviewed a single glass slide, which appeared to contain tissue from a lymph node; he reported that it was largely replaced by poorly differentiated squamous cell carcinoma, and that there was no black pigment or birefringent crystals of silica in the lymph node tissue. Dr. Naeye concluded that there was no evidence of pneumoconiosis in the very limited tissue he had available for microscopic review. Dr. Koenig

[12] Dr. Binns read the x-ray for quality purposes only.

felt that this statement was true, but very misleading since it was based on very limited tissue from the left hilar area, and ignored the clinical history, old x-rays, and results from the biopsy of the right upper lobe mass. According to Dr. Koenig:

> It falsely implies that all other large opacities, including the right upper lobe opacity, are secondary to malignancy rather than complicated CWP. As indicated above, such an assertion is incorrect since the CT guided biopsy of the right upper lobe lesion was perfectly consistent with complicated CWP and not malignancy or tuberculosis. Moreover, the left hilar abnormality was new and suspicious for a process other than complicated CWP. It is therefore not surprising that only cancer and no evidence of complicated CWP was found in this biopsy.

Dr. Koenig concluded that Mr. A. had simple coal workers' pneumoconiosis, which progressed to complicated pneumoconiosis, as confirmed by his clinical history, serial x-rays over the years, and CT guided lung biopsy of the right upper lobe large opacity. Mr. A. then developed a new left hilar mass on the background of complicated pneumoconiosis. An open lung biopsy showed that this mass was secondary to poorly differentiated squamous cell carcinoma of the lung. Dr. Koenig indicated that the results of the PET scan were perfectly consistent with the development of lung cancer on a background of complicated pneumoconiosis.

Dr. Koenig testified by deposition on October 27, 2006 (LMCX 8). He stated that it was fairly typical for coal workers' pneumoconiosis to involve the peripheral area of the lungs, and as the disease progresses, to move medially; it can also involve the pleura. In addition, as the nodules coalesce and surrounding emphysema develops it is not uncommon for the profusion of small nodules to appear to diminish. Dr. Koenig stated that tuberculosis presents in the apical portions of the lung, but complicated pneumoconiosis can as well, in addition to fungal infections.

Dr. Everett F. Oesterling

Dr. Oesterling examined medical records, and a tissue slide, at the Employer's request, and prepared a report dated June 22, 2006 (LMEX 2). On his examination of the tissue slide obtained on April 3, 2003, Dr. Oesterling noted the absence of anything resembling anthracotic pigment.[13] He described it as a solid mass, with nests of invading tumor cells, surrounded by purple infiltrate. Based on this limited tissue sample, he agreed with Dr. Estalilla's diagnosis of poorly differentiated squamous cell carcinoma with necrosis and sclerosis, and a moderate lymphocytic tumor response.

Dr. Oesterling felt that the relevant issue in this case was any potential association between exposure to coal mine dust and the evolution of this malignant tumor. He referred to Pathology of the Lung, which indicates that most studies of coal miners show that lung cancer is slightly less common in coal miners than comparable populations, and that most lung cancers in coal miners can be accounted for by the effects of cigarette smoking. He also cited to the textbook Occupational Lung Diseases, discussing the "regrettable tendency" to endorse silica as

[13] The tissue on this slide was taken during the April 3, 2003 left thoracotomy.

a carcinogen. Dr. Oesterling stated that he did not believe it was possible to associate Mr. A.'s pulmonary malignancy with his exposure to coal mine dust.

Dr. Oesterling noted that the death certificate listed the primary cause of Mr. A.'s death as lung cancer. Although it also listed complicated pneumoconiosis, Dr. Oesterling did not believe that there was any tissue confirmation of this diagnosis. But there was clear tissue documentation of Mr. A.'s primary lung cancer.

Dr. Oesterling stated that there was no lung tissue from which to determine whether coalworkers' pneumoconiosis was present. He felt that Mr. A.'s death clearly resulted from lung cancer, a condition not associated with exposure to coal mine dust. He stated that it was not possible to comment on Mr. A.'s lifetime impairment, because of the absence of functioning lung tissue, and the limited tissue that was made available. Nevertheless, he felt that it was doubtful that exposure to coal mine dust in any way produced a lifetime respiratory disability, nor would it in all probability have been a contributing factor in Mr. A.'s death. He indicated that if additional tissue became available, he would be happy to readdress these issues.

Dr. Oesterling prepared a report dated October 13, 2006, after viewing two additional tissue slides (LMEX 14).[14] He prepared photographs of the first slide, noting that on several of them, there were infrequent small birefringent silica crystals, and modest quantities of dust. Based on his tissue examination, Dr. Oesterling found evidence of a very mild anthracotic pigmentation involving the pleural surface, and a suggestion of pleural fibrosis. He stated that typically, the pleura responding to any irritant will produce reactive fibrotic change. According to Dr. Oesterling, the limited quantities of coal mine dust indicated that Mr. A. had inhaled very modest quantities of coal dust, because typically inhaled dust tends to concentrate within the pleural surface, due to its rich vascularity. Thus, the minimal pigment suggested very minimal inhaled dust.

On the second tissue slide, Dr. Oesterling noted a cluster of cells that appeared to be poorly differentiated malignant tumor cells. He felt that this slide was consistent with his previous diagnosis of poorly differentiated squamous cell carcinoma.

Dr. Oesterling felt that the limited change he saw in Mr. A.'s lung tissue was not sufficient to warrant a diagnosis of coal workers' pneumoconiosis. There was only anthracotic pigmentation of the pleural membranes with reactive fibrosis, and the mass that was interpreted clinically as conglomerate pneumoconiosis appeared to be the original excised tumor.[15] Thus, there was no tissue confirmation that Mr. A. had significant pneumoconiosis.

Dr. Paul S. Wheeler

[14] Dr. Oesterling's report does not indicate the date on which these tissue slides were made, or what part of Mr. A.'s lungs they were from, but it appears that the first was from the March 31, 2003 core biopsy of the mass in Mr. A.'s right lung, and the second was from Mr. A.'s left lung.

[15] In fact the multiple bilateral masses in Mr. A.'s lungs – not just the "original excised tumor" that was excised during the left thoracotomy - were consistently interpreted clinically as conglomerate pneumoconiosis.

Dr. Wheeler was provided with Dr. Koenig's September 29, 2006 report, and prepared a report dated October 2, 2006 (LMEX 11). Dr. Wheeler discussed his credentials at length. He stated that his experience with pneumoconiosis has shown that it produces small nodular infiltrates symmetrically in the central portion of the mid and upper lungs, and not in the periphery, and certainly not in the pleura, which has no alveoli. On the other hand, tuberculosis generally results in asymmetrical infiltrates in the apices, posterior upper lobes, and superior segments of the lower lobes, before advancing to involve other portions of the lungs. It commonly involves the pleura, and is one of the very few diseases to involve the apices.

According to Dr. Wheeler, the large opacities of pneumoconiosis develop where the small nodules predominate, which is in the central portion of the mid and upper lungs, often near the hila. They are typically bilateral, and as they progress, the small nodules merge into them, but they almost never completely disappear. In Mr. A.'s case, the February 26, 2003 CT scan showed not only a mass that involved the right and lower left apex, but it also contained calcified granulomata and involving the pleura. He stated that calcified granulomata are most commonly caused by histoplasmosis, and secondly by tuberculosis that has healed; pneumoconiosis does not cause calcified granulomata. He noted that the CT scan did not show any sign of small background nodules. Dr. Wheeler stated that the only time he has seen large opacities completely merge into all small background nodules was in a case of a glass etcher who worked unprotected over many years.

Dr. Wheeler stressed that any significant lung disease requires accurate diagnosis to assure proper therapy; that typically involves lung biopsy or positive microbiology. He stated that the diagnosis could have been made in any properly equipped hospital or clinic while Mr. A. was alive. After he died, an autopsy should have been performed to determine the cause of his death. He stated that

> Since different diseases can cause patterns identical to various pneumoconioses, histology is the most accurate way to diagnose any pneumoconiosis and should have been available to support a claim of complex coal workers' pneumoconiosis.

Dr. Wheeler testified by deposition on October 30, 2006, after reviewing a series of x-rays dating from 1997 to 2003 (LMEX 17). He stated that there was a large mass in both Mr. A.'s right and the left lung. Dr. Wheeler indicated that by the time of the December 30, 2002 x-ray, there was a 9 centimeter mass in the right upper lobe, a five centimeter mass or infiltrate in the lateral left upper lung, a two to three centimeter mass or fibrosis in the left apex, and a three centimeter mass in the lateral right mid lung. On the September 24, 2003 x-ray, he thought that there could be some small nodules of 0/1 profusion, but there were plenty of other diseases that could explain all of the findings. He did not find large opacities; rather, he felt that the masses he saw that were larger than a centimeter were conglomerate granulomatous disease, and that by location, tuberculosis would be an excellent explanation for all of them. According to Dr. Wheeler, Mr. A. could have had tuberculosis without his knowledge; it can self cure, but when it is this advanced, and causing masses of this size, it usually requires some sort of therapeutics. If there were none, and no diagnosis of tuberculosis, by exclusion it was probably histoplasmosis. Dr. Wheeler stated that if the masses were not due to tuberculosis or histoplasmosis, they could be due to metastatic disease, or sarcoid.

Dr. Wheeler felt that the x-rays he saw were not consistent with a finding of complicated pneumoconiosis, because he did not see background nodules on any of the original examinations. There was also a CT scan that did not show any background nodules; he indicated that on the x-ray, he "wouldn't have been able to tell." Dr. Wheeler testified that he has not seen large opacities that involve the apex or pleura; he likes to see symmetrical large opacities in the central portion of the mid and upper lung, surrounded by small nodules, before he is convinced that there is complicated pneumoconiosis.

Dr. Wheeler stated that at the academic centers where he has practiced, a case such as Mr. A.'s would not be allowed to go long without histologic diagnosis of the masses by direct needle biopsy. He stated "That's the way medicine should be practiced." When it was pointed out that a needle biopsy was in fact performed on Mr. A.'s right lung in March 2003, which showed the presence of anthracotic pigment, Dr. Wheeler stated that "all of us have anthracotic pigments." He indicated that the presence of anthracotic pigment on the biopsy was not enough to diagnose the presence of pneumoconiosis. According to Dr. Wheeler, "The diagnosis of pneumoconiosis is –it's a histologic diagnosis. The histology of coal workers' pneumoconiosis is distinct from the histology of silicosis, but the radiographic pattern of the nodules is identical." Dr. Wheeler testified that

> Large opacities are made up of, of coalescence of the small nodules of silicosis or coal workers' pneumoconiosis, and so they will have a distinctive histologic pattern that's different, from, say an abscess or a cancer or active granulomatous disease. The problem comes, as I understand it, with the healed granulomatous disease. Healed granulomatous disease can have a lot of fibrosis, and the silicotic and coal workers' pneumoconiosis large opacities have a lot of fibrosis all merged together. So it's - - there can be a problem when you're dealing with advanced granulomatous, or advanced healed granulomatous disease and distinguishing it from large opacities of pneumoconiosis.

Dr. Wheeler reviewed the February 26, 2003 CT scan. He testified that CT scan is the medically accepted gold standard for detecting any lung disease. He found this CT scan to show probable healed granulomatous disease, compatible with tuberculosis or histoplasmosis, with a mass in the posterior right upper lobe and lower right apex, in the lower posterior right upper lobe, and a small mass in the left apex; all contained calcified granulomata and involved the pleura. According to Dr. Wheeler, there is no reason for a large opacity of pneumoconiosis to involve the pleura, which does not have alveoli. The presence of calcified granulomata was an indication that there was a healed granulomatous process, of which tuberculosis and histoplasmosis are the two most common. There was a three centimeter mass in the lower lateral left hilum compatible with adenopathy or cancer. There were small calcified granulomata more likely than tumors involving the pleura in both lower lobes, and small calcified granulomata in the right upper hilum; there was also minimal emphysema, with areas of decreased upper lung markings. But he did not find the background nodules he likes to see with any mass that is a large opacity of coal workers' pneumoconiosis or silicosis.

Dr. Wheeler testified that he did not like the term "progressive massive fibrosis," stating that masses in the lungs usually cannot be called massive unless they take up the entire lung and totally opacity it. In his opinion, this term is archaic and misleading.

Dr. Wheeler stated that the x-ray and CT scans fit with a diagnosis of granulomatous disease. In addition, the biopsy fits, because even though it had anthracosis in it, it "undoubtedly" had other things, including fibrosis.

Dr. Wheeler was asked about the report by Dr. Koenig, who disputed his findings, and he was specifically asked if he had any medical literature to support his opinion that masses of complicated pneumoconiosis are not found in the pleura. Dr. Wheeler stated:

> I don't think I need medical literature. The coal workers' pneumoconiosis starts as small nodules almost always in the central portion of the mid and upper lungs. For a nodule of silicosis and coal workers' pneumoconiosis to form in the periphery requires extensive central involvement of the lungs. It's not going to ever form – the nodules are never going to form in the pleura because there are no alveoli in the pleura. Pleura are the fibrous tissue that lines and strengthens the surface of the lungs and lines the interior surface of the chest. So the vast majority of small - - of large opacities form in the central portion of the mid and upper lungs near the hilum. They do not involve the pleura because there are no alveoli. It's just that simple.

Dr. Wheeler stated that pulmonologists spend a small minority of their time reviewing x-rays and CT scans, and that on his worst day, he will see more x-rays and CT scans than the busiest pulmonologist in the world sees on his best day.

Dr. Wheeler acknowledged that he did not examine Mr. A. or review any of his treatment records. He also conceded that radiologists can be helped by clinical information if it is accurate. He testified that in general, he would expect to see night sweats with tuberculosis, but that was not necessary. Dr. Wheeler agreed that the mass he saw in Mr. A.'s right upper lobe remained relatively stable, which tended to favor a benign process. Dr. Wheeler testified that he was not provided with the results of the CT guided needle biopsy taken on March 31, 2003 from the mass in the upper lobe of Mr. A.'s right lung.

Death Certificate

Dr. Stanley completed Mr. A.'s death certificate, indicating that the immediate cause of his death was lung cancer, with complicated pneumoconiosis being an underlying cause (LMDX 56). He also listed as other significant conditions hypertension and degenerative disc disease.

DISCUSSION

The instant claim is a "duplicative" or "subsequent" claim because a prior claim was finally denied over one year ago. There is, accordingly, a threshold issue as to whether there are

grounds for reopening the claim under 20 C.F.R. §725.309. A subsequent claim will be denied unless the claimant can demonstrate that at least one of the conditions of entitlement upon which the prior claim was denied ("applicable condition of entitlement") has changed and is now present.[16] 20 C.F.R. §§725.309(d)(2), (3). If a claimant does demonstrate a change in one of the applicable conditions of entitlement, then generally findings made in the prior claim(s) are not binding on the parties. 20 C.F.R. §725.309(d)(4). Consequently, the relevant inquiry in a subsequent claim is whether evidence developed since the prior adjudication would now support a finding of a previously denied condition of entitlement.

In the Director's May 22, 1979 determination, Mr. A.'s claim was denied because the Director found that he did not establish that he had pneumoconiosis, or that he was totally disabled due to pneumoconiosis. Thus, Mrs. A. must establish one of these elements of entitlement in order for a consideration of the claim on the merits.

Existence of Pneumoconiosis

Section § 718.202 provides four means by which pneumoconiosis may be established. Under § 718.202(a)(1), a finding of pneumoconiosis may be made on the basis of the x-ray evidence. In this case, there are eight ILO interpretations of three x-rays taken between 1978 and 2002. The first x-ray was performed on August 1, 1997, and was read by Dr. Alexander, who is dually qualified, as showing pneumoconiosis 2/2, q, p, with category B opacities. In contrast, Dr. Wheeler, who is also dually qualified, read this x-ray as negative for pneumoconiosis.

The next x-ray, performed on September 15, 1998, was also read by Dr. Alexander to show pneumoconiosis 2/2, q, p, with category B opacities. Dr. Scott, who is dually qualified, read this x-ray as negative for pneumoconiosis.

The next x-ray was performed on December 30, 2002, and it was read as positive for pneumoconiosis 2/2, q, p, with category B opacities by Dr. Alexander. Dr. Patel, who is dually qualified, read this x-ray as positive for pneumoconiosis, 2/3, t, s, with category A opacities. However, Dr. Wheeler read this x-ray as negative for pneumoconiosis.

Thus, there are four positive interpretations by dually qualified physicians, and three negative interpretations by dually qualified physicians. Based on the preponderance of positive ILO interpretations by dually qualified physicians, I find that Mrs. A. has established by a preponderance of the x-ray evidence that Mr. A. had pneumoconiosis.

[16] For a miner, the conditions of entitlement include whether the individual (1) is a miner as defined in this section; (2) has met the requirements for entitlement to benefits by establishing pneumoconiosis, its causal relationship to coal mine employment, total disability, and contribution by the pneumoconiosis to the total disability; and (3) has filed a claim for benefits in accordance with this part. 20 C.F.R. §725.202(d) (*Conditions of entitlement: miner*).

In addition, the record includes a number of narrative reports on x-rays done at the request of Mr. A.'s physicians. Thus, Dr. Stanley's records include a report on a September 15, 1998 x-ray that was read by Dr. Cruz, who noted no significant change since an August 1997 study. Dr. Cruz described numerous small ill defined opacities again seen throughout both lungs, with some patchy densities at the upper lung fields, consistent with pneumoconiosis complicated with bilateral pulmonary fibrosis.

Dr. McCormick requested an x-ray on February 5, 2003, which was read by Dr. Willis. Dr. Willis reported a background of small parenchymal opacities consistent with a history of occupational pneumoconiosis, as well as larger masses in the right upper lobe and left mid lung zone, and a circumscribed mass in the left hilum. Dr. Willis felt that while all of these findings could represent pneumoconiosis, the mass that was contiguous with the hilum was unusual for a conglomerate mass of pneumoconiosis; he was concerned that this could be a neoplasm.

Dr. Skeens read an x-ray performed on March 31, 2003, noting a vague area of increased density in the left perihilar region, for which neoplasm was a consideration, as well as opacities in the right upper and left peripheral thorax, which he felt were conglomerate masses.

Dr. Stanley's records include a report on an x-ray performed on April 3, 2003, after Mr. A. underwent thoracotomy. Dr. Dameron, who read the ex-ray, noted a "redemonstration" of a right upper lobe mass. An x-ray read two days later by Dr. Elkins showed no change.

Dr. Chapman requested an x-ray on May 13, 2003. It was read by Dr. Smith, who noted chronic appearing interstitial lung markings with fibronodular changes and vague densities in the upper lobes bilaterally, most consistent with occupational pneumoconiosis. He also noted prominence of the right and left hilar shadows, and could not exclude adenopathy or recurrent neoplastic disease in a patient with a known clinical history of lung malignancy.

I find that these narrative x-ray reports support and document the positive ILO interpretations by Dr. Alexander and Dr. Patel. I rely on the preponderance of the positive ILO interpretations by dually qualified physicians, as supported by the narrative interpretations, and find that Mrs. A. has established by a preponderance of the persuasive x-ray evidence that Mr. A. suffered from pneumoconiosis.

Under § 718.202(a)(2), a finding of pneumoconiosis may be made on the basis of biopsy or autopsy evidence. There is no autopsy evidence in Mr. A.'s claim. However, after a CT scan showed a worrisome focal mass in Mr. A.'s left hilum that was not present on earlier films, Dr.

Chapman performed bronchoscopy with washings on March 25, 2003.[17] The pathology report showed no malignant cells. Dr. Chapman performed a CT-guided needle aspiration of a 2.2 cm focal mass from Mr. A.'s right lung on March 31, 2003. The pathology report on this mass showed fibrosis and anthracotic pigment, but no evidence of malignancy. However, a biopsy of a mass from Mr. A.'s left lung performed on April 3, 2003 showed poorly differentiated squamous cell carcinoma. Dr. Chapman then performed a left thoracotomy on May 19, 2003, and the biopsy showed poorly differentiated squamous cell carcinoma.

Dr. Oesterling was provided with a tissue slide from the April 3, 2003 biopsy of Mr. A.'s left lung mass, and on examination, agreed with Dr. Estalilla's (the pathologist) diagnosis of poorly differentiated squamous cell carcinoma; there was no anthracotic pigment. Dr. Oesterling talked at great length about the lack of any association between exposure to coal mine dust and the development of lung cancer. But his report did not address the question of whether there was pneumoconiosis in any other areas of Mr. A.'s lungs.

Dr. Oesterling was subsequently provided with two additional tissue slides to examine. He did not indicate in his report the date of the procedure from which these tissue slides were taken, or the area in Mr. A.'s lungs from which they came. Nevertheless, it appears from notations on the photographs that the first slide was from a core biopsy of Mr. A.'s right lung; as noted above, such a procedure was performed by Dr. Chapman on March 31, 2003. Dr. Oesterling found evidence of very mild anthracotic pigmentation, and a suggestion of pleural fibrosis. He concluded that this limited change was not sufficient to justify a diagnosis of pneumoconiosis. Dr. Oesterling's findings are entirely consistent with the original pathology report on this biopsy.

Dr. Oesterling also examined a second slide, again with no indication of the date or procedure from which it was taken. It appears from his description that this slide came from either the April 3, 2003 biopsy of the suspicious left lung mass, or the May 19, 2003 biopsy obtained after the left thoracotomy. Dr. Oesterling noted poorly differentiated clusters of malignant tumor cells. He felt that these cells were consistent with his earlier diagnosis of poorly differentiated squamous cell carcinoma. Although he acknowledged that he had limited material to examine, he concluded that there was no tissue evidence that Mr. A. had significant dust induced disease in his lungs at the time of his death.

It does not appear that Dr. Oesterling was aware that Mr. A. had a history of chronic masses on both side of his lungs, as shown consistently on x-rays and CT scans. Dr. Oesterling stated: "Thus again the mass interpreted clinically as conglomerate coalworkers' pneumoconiosis would appear to be the original excised tumor, . . ." This suggests to me that Dr. Oesterling was under the impression that the ONLY mass interpreted clinically as conglomerate pneumoconiosis was the cancerous mass from which this tissue slide was taken.

[17] Mr. A.'s CT scan showed bilateral changes consistent with fibrosis/pneumoconiosis; Dr. Chapman described the process on the right as a stable right basilar mass.

As he was provided with very limited medical evidence, Dr. Oesterling's opinions do not address the issue of whether the objective medical evidence as a whole establishes that Mr. A. had pneumoconiosis. Indeed, his isolated review of the tissue slides is consistent with the reports by Dr. Alexander and Dr. Koenig. But his opinions are directed only to limited pieces of evidence, and thus they provide no guidance on the issue of whether the medical evidence as a whole establishes that Mr. A. had pneumoconiosis.

Dr. Naeye was provided with a tissue slide from the April 7, 2003 CT guided needle biopsy, which he stated contained tissue from a lymph node. Not surprisingly, he reported no findings of coal workers' pneumoconiosis. Although he did not provide the basis for his assumption, Dr. Naeye then went on to state that Mr. A. "apparently" had inoperable squamous cell carcinoma in both lungs which was the presumed consequence of his heavy smoking.

In his September 11, 2006 report, Dr. Naeye addressed the comments by Dr. Alexander, stating that a diagnosis of complicated pneumoconiosis cannot be made on x-ray alone, and must be confirmed by direct tissue examination. He felt that in Mr. A.'s case, there was no such tissue confirmation. When he was subsequently provided with two tissue slides, Dr. Naeye described a small amount of black pigment, and a small number of tiny birefringent crystals.[18] He indicated that there were no lesions of pneumoconiosis.

I find that Dr. Naeye's reports do not add anything of substance to the analysis in this case. Mr. A.'s treating physicians, as well as every other physician who considered or reviewed the pathology reports, agreed that he had a cancerous mass in his left lung. Thus, Dr. Naeye's conclusion that one of the tissue slides from this mass showed cancer is hardly surprising. But Dr. Naeye's statement that Mr. A. "apparently" had cancer in both sides of his lungs has no basis in the medical evidence, nor did Dr. Naeye indicate the source of his assumption.

Dr. Naeye's analysis of the slide from the core biopsy is not inconsistent with the evaluations by the other pathologists who have examined the slide.[19] Nor has any other physician suggested that the findings on this biopsy, standing alone, support a diagnosis of coal workers' pneumoconiosis. Rather, Dr. Alexander and Dr. Koenig have pointed to the findings of anthracosis and fibrosis as supporting their conclusion, based on the totality of the medical evidence, that Mr. A. had pneumoconiosis.

Although, as discussed below, the identification of anthracosis and fibrosis on the tissue obtained from the CT guided biopsy of Mr. A.'s right lung mass is an important factor in the

[18] Although he did not indicate the date or the procedure from which these slides came, the first slide, which contained smeared exfoliated cells, appears to be from the bronchial washing of Mr. A.'s left lung performed by Dr. Chapman on March 25, 2003, and the second slide appears to be from the core biopsy performed on a mass in Mr. A.'s right lung by Dr. Chapman on March 31, 2003.

[19] Dr. Naeye stated that there was no fibrosis specifically associated with the birefringent crystals; he did not indicate that there was no fibrosis.

consideration of the totality of the medical evidence, I find that it is not sufficient, standing alone, to support a finding of pneumoconiosis based on the biopsy evidence. Thus, I find that Mrs. A. has not established pneumoconiosis by virtue of the biopsy evidence.[20]

Under § 718.202(a)(4), Mrs. A. can also establish that Mr. A. suffered from pneumoconiosis by well-reasoned, well-documented medical reports. A "documented" opinion is one that sets forth the clinical findings, observations, facts and other data on which the physician based the diagnosis. *Fields v. Island Creek Coal Co.,* 10 B.L.R. 1-19 (1987). An opinion may be adequately documented if it is based on items such as a physical examination, symptoms, and the patient's history. *See,* 20 C.F.R. § 718.107, *Hoffman v. B&G Construction Co.,* 8 B.L.R. 1-65 (1985); *Hess v. Clinchfield Coal Co.,* 7 B.L.R. 1-295 (1984). A report which is better supported by the objective medical evidence of record may be accorded greater probative value. *Minnich v. Pagnotti Enterprises, Inc.,* 9 B.L.R. 1-89, 1-90 n.1 (1986); *Wetzel v. Director, OWCP,*8 B.L.R. 1-139 (1985).

A "reasoned" opinion is one in which the administrative law judge finds the underlying documentation adequate to support the physician's conclusions. *Fields, supra.* Indeed, whether a medical report is sufficiently documented and reasoned is for the administrative law judge as the finder of fact to decide. *Clark v. Karst-Robbins Coal Co.,* 12 B.L.R. 1-149 (1989)(en banc). Moreover, statutory pneumoconiosis is established by well-reasoned medical reports which support a finding that the miner's pulmonary or respiratory condition is significantly related to or substantially aggravated by coal dust exposure. *Wilburn v. Director, OWCP,* 11 B.L.R. 1-135 (1988). An equivocal opinion, however, may be given little weight. *Justice v. Island Creek Coal Co.,* 11 B.L.R. 1-91 (1988); *Snorton v. Zeigler Coal Co.,* 9 B.L.R. 1-106 (1986).

Dr. Rasmussen, who examined Mr. A. at the Director's request, and administered objective testing, concluded that he had pneumoconiosis category A, based on his x-ray findings and his 33 year history of coal mine employment; he also concluded that he had chronic bronchitis due to his exposure to coal mine dust and cigarette smoking. I find that Dr. Rasmussen's conclusions are well-reasoned and supported by the objective medical evidence, and I accord them significant weight.

Dr. Alexander reviewed Mr. A.'s medical records, including a number of x-ray and CT scan films. He read Mr. A.'s x-rays, which spanned a period of six years, sequentially in order to make an accurate determination of which abnormalities were chronic and stable, and which were new or acute. On all of these x-rays, he found a background of small round opacities in all six lung zones that were consistent with pneumoconiosis, with areas of coalescence in the upper zones. They were stable and unchanged. Additionally, all of the x-rays showed large opacities consistent with complicated pneumoconiosis in both upper and mid zones, more extensive on the

[20] At the hearing, the Claimant requested additional time to obtain a review of the March 31, 2003 biopsy, but did not submit such a review post-hearing. However, the fact that the Claimant ultimately decided not to develop or submit such evidence, standing alone, does not justify an adverse inference that such a review was, or would be, unfavorable to the Claimant.

right, that were stable and unchanged. Dr. Alexander also noted that a left intrahilar mass appeared on the December 30, 2002 x-ray, a new and abnormal finding that was also seen on subsequent x-rays. The left thoracotomy performed on April 3, 2003 confirmed that this was a poorly differentiated squamous cell lung cancer.

Dr. Alexander reviewed Mr. A.'s chest CT scan done on February 26, 2003, noting innumerable tiny round densities bilaterally, consistent with a background of pneumoconiosis, as well as bilateral partially calcified large opacities consistent with the progressive fibrosis of complicated pneumoconiosis. He felt that the radiographic appearance of the masses was much more typical of pneumoconiotic conglomerate fibrosis than granulomatous disease, which produces smooth and regular borders, and solitary calcified nodules, of which there were none.

Dr. Alexander relied on the March 31, 2003 needle biopsy of the right upper lobe mass, which showed fibrosis and anthracotic pigment, but no evidence of malignancy. According to Dr. Alexander, the presence of carbon pigment in the mass indicated the inhalation of coal dust, and thus pneumoconiosis as the cause of the mass.

Dr. Alexander relied on the March 24, 2003 PET scan, which showed an area of mild to moderate uptake in the right upper lung, corresponding to the location of the dominant large opacity seen on x-rays and CT scan. There was also an area of significantly increased uptake in the left hilum, corresponding in size and location to the mass seen on x-rays and CT scan that was subsequently proven to be a poorly differentiated squamous cell cancer.

Dr. Alexander concluded that Mr. A. suffered from two simultaneous and independent disease processes. He had complicated pneumoconiosis, with bilateral conglomerate masses of progressive massive fibrosis, and sometime between 1998 and 2002, he developed a lung cancer adjacent to the left hilum.

Dr. Koenig, who reviewed Mr. A.'s medical records, noted that the majority of physicians, including Dr. Zaldivar, agreed that Mr. A. had radiographic evidence of simple pneumoconiosis, and several bilateral upper lobe opacities. All of the physicians agreed that the cause of the left hilar mass was cancer. But there was dispute about the cause of the other large opacities. After reviewing all of the records, Dr. Koenig determined that Mr. A. developed lung cancer, which was the cause of his left hilar mass, on a background of complicated pneumoconiosis. He pointed to the fact that these masses, other than the left hilar mass, had changed little over more than six years. In contrast, if these masses were due to lung cancer or tuberculosis, without treatment, significant progression and enlargement would have occurred. Indeed, it was the rapid appearance of the left hilar mass that alerted Mr. A.'s physicians to the possibility of cancer.

Dr. Koenig also relied on the results of the CT guided lung biopsy done on March 31, 2003, noting that if cancer is the cause of a lung mass, the likelihood of a biopsy finding cancer is very high. Yet this biopsy was negative, with findings of lung fibrosis and anthracotic pigment, exactly what would be expected if one biopsied complicated pneumoconiosis. Additionally, Dr. Koenig pointed to the lack of systemic symptoms before the appearance of the left hilar mass, which indicated that complicated pneumoconiosis was the cause of the other large upper lobe opacities, rather than cancer or tuberculosis, which produce clinical symptoms. Finally, Dr. Koenig pointed to the results of the PET scan, which produced particularly intense hypermetabolic uptake in the left hilar lymph node region, in the area that was later proven to be cancer.

Dr. Zaldivar examined Mr. A. at the Employer's request, and performed x-ray, pulmonary function, and arterial blood gas studies. But although Dr. Zaldivar is a B reader, there is no x-ray interpretation by Dr. Zaldivar attached to his report. Instead, there is an interpretation done by Dr. Wheeler, after Dr. Zaldivar prepared his report. Dr. Zaldivar discussed the pulmonary function studies he performed, and attached the results of these and the arterial blood gas studies. But he did not discuss his x-ray, or mention the results. Dr. Zaldivar also reviewed medical records provided to him by the Employer. His findings included a normal cardiopulmonary stress test, a very high carbon monoxide level of a smoker of two packs of cigarettes a day, radiological evidence of cancer in the right lung and a background of simple pneumoconiosis, according to the records he reviewed, moderate irreversible airway obstruction, airtrapping by lung volumes with hyperinflation, and normal diffusion capacity.[21]

Dr. Wheeler reviewed Dr. Koenig's report, as well as a series of x-rays dating from 1997 to 2003, and CT scans. He relied on his experience with pneumoconiosis, which has shown that it produces small nodular infiltrates symmetrically in the central portion of the mid and upper lungs, and not in the periphery, and certainly not in the pleura. In his experience, the large opacities of pneumoconiosis develop on a background of small nodules, and although the small nodules merge into the large opacities, they almost never completely disappear. He noted that Mr. A.'s February 26, 2003 CT scan showed masses in the right and lower left apex; but they contained calcified granulomata and involved the pleura. According to Dr. Wheeler, calcified granulomata are most commonly caused by histoplasmosis and healed tuberculosis, but not by pneumoconiosis. Additionally, the CT scan did not show any small background nodules.

Dr. Wheeler acknowledged that there were large masses in both sides of Mr. A.'s lungs. But these were not "large opacities," rather, they were conglomerate granulomatous disease. Because of their location, he felt that tuberculosis was an excellent explanation. According to Dr. Wheeler, Mr. A. could have had tuberculosis without knowing it; however, he acknowledged that when it was this advanced, causing masses of this size, it usually required some type of therapy. If Mr. A. were not diagnosed or treated for tuberculosis, then Dr. Wheeler felt that by

[21] Dr. Zaldivar concluded that in addition to simple pneumoconiosis, Mr. A. had cancer that had the radiologic appearance of complicated pneumoconiosis in his right lung. Dr. Zaldivar's opinions on this issue are discussed further below.

exclusion it was "probably" histoplasmosis. But if not, the masses could be due to metastatic disease or sarcoid.

It appears that Dr. Wheeler was not provided with the results of the various biopsies performed on Mr. A.'s lungs. This would explain his admonishment that, at the places where he practiced, Mr. A.'s case would not be allowed to go long without histologic diagnosis of the masses by direct needle biopsy, and "That's the way medicine should be practiced." However, when it was pointed out to Dr. Wheeler that the March 2003 needle biopsy of Mr. A.'s right lung showed the presence of anthracotic pigment, he brushed it off by stating that "all of us have anthracotic pigments," stating that the presence of anthracotic pigment was not enough to diagnose the presence of pneumoconiosis. He would not answer repeated questions about whether those findings were *consistent with* pneumoconiosis, stating that the diagnosis of coal workers' pneumoconiosis was histologic, and distinct from the histology of silicosis.[22] He felt that the x-ray and CT scans fit with a diagnosis of granulomatous disease, as did the biopsy, speculating that even though it showed anthracosis, it "undoubtedly" had other things, including fibrosis. Dr. Wheeler based much of his conclusions on his experience that masses of complicated pneumoconiosis are not found in the pleura. Yet when he was asked if he could support that opinion with medical literature, he stated that he did not need medical literature.

The focus of Dr. Wheeler's testimony was on the etiology of the large masses in Mr. A.'s lungs. Thus, he did not directly address the issue of the presence of simple pneumoconiosis, other than to repeat that he expected to see a background of simple pneumoconiosis with complicated pneumoconiosis, which he did not see. But more important, the reasonable inference to be drawn from Dr. Wheeler's report and testimony is that he does not accept a diagnosis of pneumoconiosis based on x-ray or CT scan alone; rather, he must see positive microbiology from a lung biopsy or autopsy. Clearly, he felt that Mr. A.'s care providers were not competent to make such a diagnosis, and that an autopsy should have been performed after he died to determine the precise cause of his death. However, the statute and regulations do not require Mrs. A. to satisfy Dr. Wheeler's diagnostic criteria before she is entitled to benefits.

Nor was Dr. Wheeler familiar with Mr. A.'s medical history, including the reports from his treating physicians that reflect a consistent history of x-ray findings of pneumoconiosis, and in the last few years of his life, the development of a cancerous tumor on a background of complicated pneumoconiosis. Mr. A. had no history of tuberculosis, or indication of the systemic symptoms that accompany tuberculosis. I find that Dr. Wheeler's opinions are based on an incomplete consideration of the available medical evidence, and I accord them little, if any weight.

Dr. Alexander and Dr. Koenig are the only physicians who have addressed the totality of the medical evidence of record. I find that their opinions are eminently well-reasoned, as well as supported by the objective medical evidence of record. They incorporate all of the relevant

[22] Of course, under the statute and regulations, "silicosis" is synonymous with pneumoconiosis.

medical findings, including x-ray and CT scan evidence, biopsy evidence, PET scan evidence, and clinical history. In contrast, Dr. Zaldivar, Dr. Naeye, Dr. Oesterling, and Dr. Wheeler addressed selected medical evidence, without taking into account highly relevant findings. They were provided with only limited and selective pieces of a much larger puzzle. Dr. Koenig, who did cite to medical literature to support his opinions, stated that Dr. Wheeler's opinions were not supported by, and indeed were contrary to, the medical literature. Dr. Wheeler confirmed that he does not need to rely on medical literature. However, I am not willing to rely on a physician whose opinions are based on an incomplete consideration of the medical records, are at odds with the opinions of physicians who have considered the complete medical record, and who does not see the need to support his opinions with any medical literature or studies.

I find that the reports by Dr. Zaldivar, Dr. Naeye, Dr. Oesterling, and Dr. Wheeler are based on a woefully inadequate consideration of the medical evidence, which renders them unpersuasive and unreliable. I accord them little, if any, weight. Relying on the abundantly well-reasoned opinions of Dr. Alexander and Dr. Koenig, as supported by the report of Dr. Rasmussen, Mr. A.'s medical records, and the results of objective testing, I find that Mrs. A. has established the existence of pneumoconiosis by the overwhelming preponderance of the medical opinion evidence.

Section 718.202(a)(3) provides that pneumoconiosis may be established if any one of several cited presumptions are found applicable. In the instant case, the presumption of § 718.305 does not apply to claims filed after January 1, 1982. Section 718.306 does not apply to claims where the miner died after March 1, 1978. Section 718.304 allows a presumption of complicated pneumoconiosis where, *inter alia*, an x-ray "yields one or more large opacities (greater than 1 centimeter in diameter) and would be classified in Category A, B, or C" if such miner is suffering or suffered from a chronic dust disease of the lung. 20 C.F.R. § 718.304(a). However, if the employer can affirmatively show the opacity is something other than pneumoconiosis, the x-ray loses force, and the claimant loses the benefit of the presumption. *See Eastern Associated Coal Corp. v. Director, OWCP [Scarbro]*, 220 F.3d 250, 256 (4th Cir. 2000).

In this case, there is no disagreement about the presence of large masses in both sides of Mr. A.'s lungs. Thus, Dr. Patel and Dr. Alexander have designated these masses as category A or B opacities on x-ray. Although he did not designate them as category A, B, or C opacities, Dr. Scott described significant processes on Mr. A.'s x-ray. Likewise, Dr. Wheeler did not designate the masses he acknowledged were on x-ray as category A, B, or C opacities. These masses were also described on CT by every physician who examined the CT scans. Nor is there any disagreement that Mr. A. had a cancerous mass in his left lung, as shown on biopsy and PET scan.

The issue is the etiology of the large and stable masses that appeared on Mr. A.'s x-rays from 1997 onward. Again, I place greatest reliance on the opinions of Dr. Alexander and Dr. Koenig, who reviewed all of the medical evidence, and determined that at the time of his death, Mr. A. suffered from a left lung cancer superimposed on a background of bilateral complicated

pneumoconiosis. Their opinions are well-reasoned, and more than adequately supported by the objective medical evidence. They relied on the radiographic appearance of the masses, as being more typical of pneumoconiotic conglomerate fibrosis than granulomatous disease, on the results of the March 31, 2003 needle biopsy of the right upper lobe mass, which showed fibrosis and anthracotic pigment, on the results of the PET scan, which showed a lesser degree of uptake in the right masses, and the absence of systemic symptoms that would suggest tuberculosis.

In contrast, I find that the Employer has not met its burden to affirmatively show that the masses that appear on x-ray as category A and B opacities are due to a disease process other than pneumoconiosis. Thus, Dr. Naeye's opinion was limited to his finding that there were no lesions of coal workers' pneumoconiosis in the tissue slides he examined. One of these slides came from Mr. A.'s cancerous tumor, one from a bronchial washing, and on the third, Dr. Naeye reported black pigmentation with a few tiny birefringent crystals. Dr. Naeye's focus was extremely narrow; he did not address the etiology of the large masses that were unanimously acknowledged to be present, not only in Mr. A.'s left lung, but in his right lung as well. In fact, Dr. Naeye's finding of pigment and birefringent crystals on the slide from Mr. A.'s right lung is entirely consistent with the reports by Dr. Alexander and Dr. Koenig. Thus, I find that Dr. Naeye's opinions are not helpful, as they do not address the totality of the medical evidence, and I have accorded them little, if any, weight.[23]

Dr. Zaldivar acknowledged that Mr. A.'s x-ray had the radiographic appearance of complicated pneumoconiosis in his right lung. But he felt that Mr. A.'s history "clearly" showed that this mass was due to cancer that had spread, according to the PET scan. But Dr. Zaldivar was not aware that the masses in Mr. A.'s lungs predated the development of the cancerous tumor by a number of years. Nor was he aware of the core biopsy that was performed on one of the masses in Mr. A.'s right lung seven days after the PET scan, which had no findings of malignancy. According to Dr. Koenig, if there were cancer in this mass, it would have shown up in the core biopsy. Nor was Dr. Zaldivar aware of the left thoracotomy, which showed the tumorous mass, as well as nodules of metastasis, not in the location or to the extent indicated on the PET scan. According to Dr. Alexander and Dr. Koenig, the findings on PET scan were in fact consistent with the appearance of complicated pneumoconiosis, which can cause increased uptake; in contrast, the uptake on the cancerous tumor was 100% brighter. Dr. Koenig noted that even Dr. Smith, who prepared the PET scan report, acknowledged the possibility that complicated pneumoconiosis could be causing some of the areas of uptake, and recommended CT guided biopsy of the right upper lung mass to resolve this possibility. The subsequent CT guided biopsy, in fact, contained anthracotic pigment, but no malignancy. But most importantly, Dr. Zaldivar saw only a limited piece of the available medical evidence. As his opinions are based on an incomplete consideration of the medical evidence, I find that Dr. Zaldivar's conclusions are not entitled to any significant weight.

[23] In addition, Dr. Naeye's opinion that complicated pneumoconiosis cannot be diagnosed with x-ray findings alone, but must be confirmed by tissue examination, is inconsistent with the statutory scheme, which does not require a medical diagnosis of complicated pneumoconiosis, and provides for application of the irrebuttable presumption of total disability due to pneumoconiosis based on x-ray evidence alone.

Apparently under the impression that the "relevant issue" in this case was whether the malignant tumor he identified on the slide from Mr. A.'s left lung was associated with his exposure to coal mine dust, Dr. Oesterling discussed studies showing no association between the exposure to coal mine dust and the development of lung cancer. When he was later asked to examine a slide that apparently was from Mr. A.'s right lung core biopsy, as well as an additional slide apparently from the cancerous tumor on the left, Dr. Oesterling found limited anthracotic pigmentation on the first slide, and malignant tumor cells on the second slide. Dr. Oesterling concluded that the limited anthracotic pigment was not sufficient to support a diagnosis of pneumoconiosis. As discussed above, no other physician concluded that, standing alone, the findings on this slide supported a diagnosis of pneumoconiosis.

But Dr. Oesterling apparently was not aware that Mr. A. had large masses on the right side of his lung: he stated that the mass that was interpreted clinically as conglomerate pneumoconiosis appeared to be the original excised tumor. Although the lack of information identifying the tissue specimens that Dr. Oesterling examined makes it somewhat difficult to understand his report, I find that it is reasonable to infer that Dr. Oesterling was under the impression that the only area of conglomerate pneumoconiosis that was diagnosed clinically was the area where the tumor was removed from his left lung, and which was subsequently determined to be cancerous. Perhaps Dr. Oesterling was under the impression that the tissue slide with anthracotic pigmentation came from the tissue surrounding this tumor. In any event, I find that, through no fault of Dr. Oesterling, who could only work with what he was provided, his report is confusing, and certainly does not establish that the large masses on the right side of Mr. A.'s lungs were due to a disease process other than pneumoconiosis.

Dr. Wheeler is the only physician to actually address the etiology of the large masses shown on x-rays and CT scans, and conclude that they were not due to pneumoconiosis, offering a variety of explanations for their development. Dr. Wheeler's review was limited to the x-rays and CT scans, as well as Dr. Koenig's report; he was not provided with any other medical records, including the results of the biopsies that he testified are so crucial for a correct diagnosis. I find that Dr. Wheeler's reasons for excluding pneumoconiosis as the etiology for the large masses that he acknowledged were present on Mr. A.'s x-rays have been addressed and successfully refuted by Dr. Alexander and Dr. Koenig.

Dr. Alexander, pulling no punches, characterized Dr. Wheeler's statement that the masses were not due to pneumoconiosis because the profusion of nodules was minimal and the masses were peripheral without pleural involvement as "fairly absurd." According to Dr. Alexander, large masses of complicated pneumoconiosis can be centrally or peripherally located, or both, and occasionally extend to the pleural surface. He also stated that it was well known that as simple pneumoconiosis progressed to complicated pneumoconiosis, with the development of large conglomerate masses, the apparent profusion of small opacities actually decreased as they were incorporated in the larger masses, and the lung parenchyma became attenuated by emphysema and thoracic distortion. Dr. Alexander stated that the radiographic appearance of the masses seen on Mr. A.'s x-rays was much more typical of pneumoconiotic conglomerate fibrosis

than granulomatous disease, which results in smooth and regular borders, and solitary calcified pulmonary nodules, of which there were none on Mr. A.'s films.

Dr. Koenig felt that not only were Dr. Wheeler's opinions unsupported, they were refuted by the medical literature. Thus, noting that Dr. Wheeler attributed the masses on x-ray to tuberculosis because the profusion of nodules was minimal and the masses were peripheral, Dr. Koenig stated that the medical literature establishes that complicated pneumoconiosis occurs most commonly in the apical posterior portions of the upper lobes, or the superior segments of the lower lobes, and starts as a mass near the periphery of the lung. Additionally, he indicated that as simple pneumoconiosis progresses to complicated, it is very common to see the apparent profusion of small opacities decrease, as a result of the small opacities being incorporated into the large opacities, and the development of emphysema and thoracic distortion. Dr. Koenig cited to numerous publications in support of his report. In contrast, when asked during his deposition whether there was medical literature to support his opinions, Dr. Wheeler stated that he did not think he needed medical literature to support his opinions.

In addition, I find that Dr. Wheeler's speculations on the etiology of the large masses in Mr. A.'s right lung are not affirmative evidence that these masses were due to a process other than pneumoconiosis. Dr. Wheeler was not provided with any of the medical records reflecting Mr. A.'s medical history, or the results of any of the biopsies conducted on both the right and left lung masses. Thus, he apparently was not aware that Mr. A. had no history of tuberculosis, or exposure to tuberculosis, or any of the systemic symptoms that come with tuberculosis. Dr. Wheeler first speculated that the conglomerate masses were probably due to tuberculosis. However, he hedged his bets, stating that in the event Mr. A. did not have the systemic symptoms that would most likely result from such an advanced case, his conglomerate masses were probably due to histoplasmosis. Or maybe cancer or sarcoid. The only thing he was sure of was that pneumoconiosis was not involved. I find that Dr. Wheeler's opinions are entitled to no weight, as they are based on an incomplete review of the medical evidence, are speculative, and are entirely supported by medical literature or studies.[24]

Finally, I note that Mr. A.'s medical records are clearly consistent with and support the conclusions of Dr. Alexander and Dr. Koenig. Mr. A.'s x-rays dating back to 1997 reflect findings of simple and complicated pneumoconiosis on both sides of his lungs. This condition was chronic and stable until late 2002, when a suspicious mass appeared on his left lung, and he was evaluated by CT scan, PET scan, bronchoscopy, core needle biopsy, and thoracotomy. After this thorough workup, Mr. A. was diagnosed with lung cancer that was found in the suspicious left lung mass, which eventually caused his death. As pointed out by Dr. Alexander and Dr. Koenig, who reviewed the totality of available medical evidence, these records establish that from at least 1997, Mr. A. had conglomerate masses of pneumoconiosis in both lungs, which

[24] Both Dr. Wheeler and Dr. Naeye believe that complicated pneumoconiosis cannot be diagnosed on the basis of x-ray alone, and that tissue confirmation is needed. However, the Act does not incorporate a purely medical definition of the condition known as complicated pneumoconiosis, and under the statutory scheme, x-ray evidence alone can be sufficient to establish entitlement to the presumption.

remained stable over many years. He then developed a cancerous tumor in his left lung, superimposed on the background of complicated pneumoconiosis.

I find that Dr. Alexander's and Dr. Koenig's opinions are persuasive and compelling, and convincingly address and refute the speculations by Dr. Zaldivar and Dr. Wheeler on the etiology of the large masses in Mr. A.'s lungs.

Relying on the opinions of Dr. Alexander and Dr. Koenig, which I find are eminently well-reasoned and supported by the objective medical evidence, I find that Mrs. A. has established that Mr. A. had pneumoconiosis by a preponderance of the x-ray evidence, by a preponderance of the medical opinion evidence, and pursuant to the presumption provided by Section 718.304. Additionally, I have considered all of the evidence as it relates to the existence of pneumoconiosis, and I find that it establishes overwhelmingly that Mr. A. suffered from pneumoconiosis. As Mr. A. worked for more than ten years as a coal miner, Mrs. A. is entitled to the regulatory presumption, which has not been rebutted, that Mr. A.'s pneumoconiosis arose from his coal mine employment.

As Mrs. A. has established that Mr. A. had pneumoconiosis, she has established a change in condition since the May 22, 1979 denial of his previous claim, and she is entitled to consideration of the claim on the merits. I note that the vast majority of medical records in connection with this claim post date the previous claim by a number of years. The limited medical evidence in the previous claim file included an x-ray report dated April 23, 1979, which was negative for pneumoconiosis. I note that the Courts have recognized that pneumoconiosis is a progressive disease. The fact that Mr. A.'s x-ray was negative in 1979 does not detract from the force of the x-ray evidence that clearly indicates that by 1997, Mr. A. had developed pneumoconiosis.

Mrs. A. must also establish that Mr. A. was totally disabled by pneumoconiosis. The arterial blood gas and pulmonary function studies that are in the record do not meet the regulatory criteria to establish total respiratory disability. The physicians who specifically addressed the issue concluded that Mr. A. had the respiratory capacity to perform his former coal mine work.

However, I have found that Mrs. A. has successfully met the requirements of Section 718.304, that is, she has established that Mr. A. suffered from the statutory condition referred to as complicated pneumoconiosis. Thus, she is entitled to the irrebuttable presumption that Mr. A. was totally disabled due to pneumoconiosis.

I find that Mrs. A. has established that Mr. A. had pneumoconiosis that arose from his coal mine employment, and that he was totally disabled due to pneumoconiosis. Accordingly, she is entitled to benefits in connection with Mr. A.'s living miner's claim.

Survivor's Claim

In connection with the survivor's claim, the parties have designated the following medical evidence.[25]

X-ray Evidence

Exhibit No.	*Date of X-ray*	*Reading Date*	*Physician/ Qualifications*	*Impression*
ESX 4	8-1-97	2-8-05	Wheeler/B, BCR	Negative for pneumoconiosis
CSX 1	8-1-97	11-10-04	Alexander/B, BCR	2/2, q, p, category B opacities
ESX 6	9-15-98	2-8-05	Wheeler/B, BCR	Negative for pneumoconiosis
CSX 1	9-15-98	11-10-04	Alexander/B, BCR	2/2, q, p, category B opacities
ESX 15	12-30-02	8-19-03	Scott/B, BCR	Negative for pneumoconiosis
CSX 1	12-30-02	11-10-04	Alexander/B, BCR	2/2, q, p, category B opacities
CSX 1	5-13-03	11-10-04	Alexander/B, BCR	2/2, q, p, category B opacities
ESX 16	5-13-03	2-8-05	Wheeler/B, BCR	Negative for pneumoconiosis

Pulmonary Function and Arterial Blood Gas Evidence

The Claimant has not designated any pulmonary function or arterial blood gas studies as evidence in connection with her survivor's claim. The Employer has designated a pulmonary function study performed by Dr. Durham on October 9, 1998 (SDX 9, 11), and arterial blood gas studies performed by Dr. Stanley on October 9, 1998 (SDX 9), and Dr. Zaldivar on September 24, 2003 (SDX 13). None of these studies produced values that meet the regulatory requirements to establish a presumption of total respiratory disability.

Medical Opinion Evidence

The Claimant has designated Dr. Alexander's November 10, 2004 report (CSX 1), and his supplemental January 10, 2005 report (CSX 2), as well as Dr. Koenig's June 15, 2005 report (SDX 14), and the transcript of his October 27, 2006 testimony (CSX 4).

[25] I have relied on the parties' evidence summary forms, as modified by respective counsel at the hearing.

The Employer has designated the transcript of Dr. Wheeler's October 30, 2006 deposition (ESX 17).

Biopsy Evidence

The Claimant has designated the report by Dr. William Mangano regarding the March 31, 2003 needle biopsy of Mr. A.'s right lung (SDX 13).

The Employer has designated Dr. Naeye's January 29, 2004 biopsy report (ESX 3), and his October 6, 2006 biopsy report (ESX 13)as initial evidence; the Employer has listed Dr. Naeye's September 11, 2006 report (ESX 12) as rehabilitation in response to Dr. Alexander's criticisms of his opinions. The Employer has also listed Dr. Oesterling's June 22, 2006 biopsy report (ESX 2) as rebuttal of the April 3, 2003 biopsy report at DX 21; and Dr. Oesterling's October 13, 2006 biopsy report (ESX 14) as rebuttal of the March 31, 2006 biopsy report at DX 21.

Other Medical Evidence

The Claimant has designated Dr. Rose's review of Mr. A.'s February 26, 2003 CT scan, found at SDX 10.

The Employer has listed Dr. Wheeler's report on the February 26, 2003 CT scan (ESX 8) as initial evidence, and Dr. Wheeler's October 2, 2006 report (ESX 11) as rehabilitation in response to Dr. Koenig's criticisms of his opinions.

Hospitalization Records and Treatment Notes

The Claimant has designated the treatment records from Summersville Memorial Hospital at SDX 9: the treatment records from the Charleston Area Medical Center at SDX 10; the treatment records from Dr. Stanley at SDX 11-12; and the treatment records from the Charleston Area Medical Center found at SDX 13.

The Employer has designated the report of the April 3, 2003 biopsy performed at the Charleston Area Medical Center (DSX 13)

DISCUSSION

The only evidence that has not been discussed above is the interpretations of the May 13, 2003 x-ray by Dr. Alexander and Dr. Wheeler, as set out in the chart above. Otherwise, I incorporate my description of the remaining evidence as set out above in the living miner's claim by reference.

Establishment of Pneumoconiosis

With respect to the x-ray evidence, I note that in the survivor's claim the interpretations are equally balanced, with four positive readings by dually qualified physicians, and four negative readings by dually qualified physicians. I find that, standing alone, the x-ray evidence is not sufficient to establish that Mr. A. had pneumoconiosis. Nor do the narrative interpretations, by physicians whose qualifications are unknown, tip the balance in favor of the Claimant. Thus, I find that Mrs. A. has not established by a preponderance of the x-ray evidence that Mr. A. had pneumoconiosis.

I also incorporate my discussion of the biopsy evidence as set out in the discussion of the living miner's claim. In other words, I find that, despite the findings of anthracotic pigment and fibrosis on evaluation of the March 31, 2003 core biopsy of Mr. A.'s right lung, the biopsy evidence, standing alone, is not sufficient to support a finding of pneumoconiosis. Thus, I find that Mrs. A. has not established by a preponderance of the x-ray evidence that Mr. A. had pneumoconiosis.

However, again relying on the reports by Dr. Alexander and Dr. Koenig, which I find to be eminently reasonable and supported by the objective medical evidence, I find that Mrs. A. has established that Mr. A. had pneumoconiosis by a preponderance of the reliable medical opinion evidence. For the same reasons as discussed in the living miner's claim, I credit the opinions of Dr. Alexander and Dr. Koenig, who reviewed and considered the totality of the available medical evidence, over the opinions of Dr. Wheeler, whose review of the medical evidence was selective. Again, weighing all of the evidence relating to the issue of whether Mr. A. had pneumoconiosis together, I find that Mrs. A. has established by an overwhelming preponderance of the evidence that Mr. A. had pneumoconiosis. As Mr. A. worked for more than ten years as a coal miner, Mrs. A. is entitled to the regulatory presumption, which has not been rebutted, that Mr. A.'s pneumoconiosis arose from his coal mine employment.

Death Due to Pneumoconiosis

To be entitled to benefits, Mrs. A. must establish that her husband's death was due to pneumoconiosis. Since the claim was filed after January 1, 1982, the issue of death due to pneumoconiosis is governed by § 718.205(c), as amended, which states, in pertinent part:

For the purpose of adjudicating survivor's claims filed on or after January 1, 1982, death will be considered to be due to pneumoconiosis if any of the following criteria is met:

(1) Where competent medical evidence establishes that pneumoconiosis was the cause of the miner's death, or

(2) Where pneumoconiosis was a substantially contributing cause or factor leading to the miner's death or where the death was caused by complications of pneumoconiosis, or

(3) Where the presumption set forth at § 718.304 is applicable.

(4) However, survivors are not eligible for benefits where the miner's death was caused by a traumatic injury or the principal cause of death was a medical condition not related to pneumoconiosis, unless the evidence establishes that pneumoconiosis was a substantially contributing cause of death.

(5) Pneumoconiosis is a "substantially contributing cause" of a miner's death if it hastens the miner's death.

20 C.F.R. § 718.205(c).

In this case, Dr. Stanley, who was one of Mr. A.'s treating physicians, completed his death certificate, indicating that the cause of his death was lung cancer, but that complicated pneumoconiosis was a contributing cause. Dr. Stanley's records reflect that he was aware of the evaluations and tests performed on Mr. A., and his course of treatment.

Neither Dr. Alexander nor Dr. Koenig directly addressed the issue of whether Mr. A.'s pneumoconiosis played a role in his death. Dr. Wheeler maintained that Mr. A. did not have pneumoconiosis, and thus by inference it could not have played a part in his death.

I find that Dr. Stanley's report on Mr. A.'s death certificate, as supported by his treatment records, is sufficient to support a finding that Mr. A.'s pneumoconiosis contributed to his death from lung cancer.

But additionally, I find that Mrs. A. is entitled to the irrebuttable presumption that Mr. A.'s death was due to pneumoconiosis. In this regard, I incorporate my discussion of the issue of complicated pneumoconiosis, as set out above in the living miner's claim. I find that Dr. Alexander's and Dr. Koenig's opinions are persuasive and compelling, and convincingly address and refute the speculations by Dr. Zaldivar and Dr. Wheeler on the etiology of the large masses in Mr. A.'s lungs. As discussed above in connection with the living miner's claim, relying on the opinions by Dr. Alexander and Dr. Koenig, I find that Mrs. A. has established by an overwhelming preponderance of the evidence that Mr. A. had the statutory condition referred to as complicated pneumoconiosis. Thus, she is entitled to the irrebuttable presumption that his death was due to pneumoconiosis.

CONCLUSION

In both the living miner's and the survivor's claims, Mrs. A. has met her burden to establish that Mr. A. had pneumoconiosis that arose from his coal mine employment. Additionally, she has met her burden to establish that Mr. A. was totally disabled due to pneumoconiosis, and that his death was due to pneumoconiosis. Accordingly, she is entitled to benefits under the Act in connection with both claims.

ORDER

Based on the foregoing, IT IS HEREBY ORDERED that the claims of M. F. A., surviving spouse of S. L. A., for black lung benefits under the Act are GRANTED.

IT IS FURTHER ORDERED that the Employer, Peerless Eagle Coal Company, shall pay to Mrs. A. all benefits to which she is entitled under the Act in connection with Mr. A.'s living miner's claim commencing in October 2002. [26]

IT IS FURTHER ORDERED that the Employer, Peerless Eagle Coal Company, shall pay to Mrs. A. all benefits to which she is entitled under the Act in connection with her survivor's claim commencing in February 2005.

A

LINDA S. CHAPMAN
Administrative Law Judge

ATTORNEY'S FEES

An application by Claimant's attorney for approval of a fee has not been received. Thirty days is hereby allowed to Claimant's counsel for submission of such an application. A service sheet showing that service has been made upon all the parties, including the claimant, must accompany the application. The parties have ten days following receipt of any such application within which to file any objections. The Act prohibits the charging of a fee in the absence of an approved application.

NOTICE OF APPEAL RIGHTS: If you are dissatisfied with the administrative law judge's decision, you may file an appeal with the Benefits Review Board ("Board"). To be timely, your appeal must be filed with the Board within thirty (30) days from the date on which the

[26] The x-ray evidence of complicated pneumoconiosis predates Mr. A.'s subsequent application, and thus I have used the date of application as the date of onset of benefits.

administrative law judge's decision is filed with the district director's office. *See* 20 C.F.R. §§ 725.458 and 725.459. The address of the Board is: Benefits Review Board, U.S. Department of Labor, P.O. Box 37601, Washington, DC 20013-7601. Your appeal is considered filed on the date it is received in the Office of the Clerk of the Board, unless the appeal is sent by mail and the Board determines that the U.S.

Postal Service postmark, or other reliable evidence establishing the mailing date, may be used. *See* 20 C.F.R. § 802.207. Once an appeal is filed, all inquiries and correspondence should be directed to the Board.

After receipt of an appeal, the Board will issue a notice to all parties acknowledging receipt of the appeal and advising them as to any further action needed.

At the time you file an appeal with the Board, you must also send a copy of the appeal letter to Donald S. Shire, Associate Solicitor, Black Lung and Longshore Legal Services, U.S. Department of Labor, 200 Constitution Ave., NW, Room N-2117, Washington, DC 20210. *See* 20 C.F.R. § 725.481.

If an appeal is not timely filed with the Board, the administrative law judge's decision becomes the final order of the Secretary of Labor pursuant to 20 C.F.R. § 725.479(a).